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THE MEDICAL DISEASES OF EGYPT

F. M. SANDWITH

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THE MEDICAL DISEASES OF EGYPT

BY

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PART I

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DEDICATED

TO MY MEDICAL COLLEAGUES IN EGYPT,
MANY OF WHOM HAVE HELPED ME TO WRITE THIS WORK,
BUT NONE OF WHOM ARE RESPONSIBLE FOR ITS
IMPERFECTIONS.

"What cannot totally be known, ought not to be totally neglected; for the knowledge of a part is better than the ignorance of the whole."

ISMAIL IBN ALI ABU EL FEDA
(Preface to his Geography.)

M351944

PREFACE

It is more than half a century since my German predecessors, Pruner and Griesinger, wrote their works on Egyptian diseases, and since that time no one has published in any language a systematic book upon medicine as seen in Egypt, though several valuable monographs have appeared relating to many individual diseases. I have therefore been invited to amplify my lectures to the students of the Egyptian Government School of Medicine, and, wishing to avoid the task of writing a complete treatise on the Principles and Practice of Medicine for which indeed I am not competent, I have assumed while writing, as in lecturing, that my readers are at least possessed of such an excellent handbook on general medicine as that of Dr. Frederick Taylor or Professor Osler.

I have therefore omitted or referred very briefly to such sections as have been fully treated by these authors, and have only written at greater length on those subjects which a very varied experience of more than 21 years in Egypt has shown to be of importance to the practitioner in that country.

I have attempted to give such information as I have been able to acquire about the history and geographical distribution of each disease in Egypt, and to furnish the reader with a list of useful references, which is, of course, not a complete bibliography, and by no means represents the number of works which I have specially consulted and sometimes culled from during the last two years.

While writing I have tried to think of the needs of three classes of possible readers—the Egyptian student of medicine, past and present—the newly appointed English doctors who are suddenly

placed in positions of responsibility in Egypt, without any knowledge of what sort of diseases they are likely to meet with among the natives—and the large and increasing number of colleagues in Europe and elsewhere, who are interested in the behaviour of medical diseases in foreign countries.

Parts of several papers now published in this first volume, have already appeared in the columns of the *Practitioner*, *Lancet*, *British Medical Journal*, *Journal of Tropical Medicine*, *Encyclopædia Medica* and other medical publications. The second volume will, I hope, complete the infectious diseases, and begin those of the nervous system.

I venture to beg readers to kindly write to me about such errors as they discover.

31 Cavendish Square, W.
1905.

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THE
MEDICAL DISEASES OF EGYPT

MEDICAL HISTORY IN EGYPT

IN pre-historic times the inhabitants of Egypt probably spent much of their time in hunting animals or in fighting among themselves, and must have been dependent on their friends who washed their wounds and dressed them with herbs. The cleverest among these doubtless became empirical medical attendants.

The earliest hieroglyph on inscriptions for a physician is *swn*, which signifies wisdom or knowledge, and is represented by an arrow with a broken shaft, because the word for arrow was phonetically the nearest to the word for wisdom. This is analogous in other languages, such as medicine from *medh*, to be wise, doctor for the teacher, and wise, wit, witch and wizard all derived from the old English *wit*, to know; again the Arabic *hakeem* has a similar etymology.

In the Medical School library in Cairo is a photographic representation I have had taken from the tomb of one of the earliest of the world's physicians, the stela of which is in the Cairo museum. This portrait dates from the Vth Dynasty (Sakkara), when surgery at least must have been well-known, for in the anatomical museum of the Cairo medical school there is an instance of a fracture of the femur, treated by four wooden splints enveloped in linen, which have been tied round the thigh with bandages. In another tomb of the Vth Dynasty (near Girga) a child was found with a fracture of the radius and ulna, the arm being tied up with splints of bark, which had

previously been wrapped round with linen. There had evidently been some hæmorrhage in this case, for a little pad of fibre had been put over a wound under the splint. [1]

The earliest known triumphs of the healing art were probably celebrated in Egypt, where the custom prevailed of laying the sick people out before their houses, so that the passers-by might tender their advice as to the treatment of their diseases. Later on, the priests included the healing art in their studies in the Temple schools such as Heliopolis, Memphis and Thebes, and believed that life should be indefinitely prolonged, unless someone or something caused death, such as a spirit, or the soul of a dead man, which cunningly entered a living person. They therefore considered that they had two equally important duties to perform, the first was to discover the spirit in possession, and then to drive it out by powerful magic, reciting incantations, and making the patient wear amulets. Diet and drugs were afterwards prescribed to counteract the disorders which the strange being had produced in the body.

It is interesting to notice this old belief, because the modern Egyptian is still unconsciously pursuing it.

Isis was the principal Egyptian deity who presided over the cure of disease, and she had the reputation of having recalled to life her son Horus; Isis and Serapis were both worshipped in Rome in later times as medical divinities, and a temple in honour of Isis existed there as late as B. C. 50. Chonsu, "the counsellor of the sick," was of lower rank, but the cat-headed Pasht and the Ape were honoured as deities of lying-in women and of fecundity, for, in all times, the possession of children has been esteemed the greatest blessing among the Egyptians. Hence the cat was sacred to Pasht, and death was the penalty for killing one; this may account, possibly, for the modern belief in Egypt that it is dangerous to ill-treat a cat, especially about sunset, for what appears to be a cat is thought to be really a spirit in disguise.

Imhetep was a king of Egypt in the IIIrd Dynasty, and according to Manetho, he, like Teta, the son of Mena, was also a physician, and wrote some works which still existed at the

[1] Both of these sets of splints were discovered by the Hearst Expedition of the University of California.

beginning of the Christian era. In course of time he was deified, and promoted to be the son of Ptah, the oldest of the gods, and Sekhet, the goddess who represented the power of the sun, and, with these two parents, formed the great triad, worshipped at Memphis. His cult increased in Saïte and Greek times, the Greeks called him Imuthes, and likened him to Æsculapius. A temple was built to him at Memphis, where the library contained manuscripts which served later on as the basis for the Ebers papyrus, and eventually inspired remedies to Galen. This temple was a resort for sick people, and similar temples in his honour were erected in other parts of Egypt, where the priests carried on the work of healing; statuettes and reliefs represent him as a young man wearing a close cap, and often with an open roll of papyrus on his knees.

The earliest medical document from Egypt or elsewhere is a papyrus dating from the XIIth dynasty, which was found at Kahûn in 1889, consisting of medical prescriptions for midwives.

Thoth, generally represented with the head of an ibis, was esteemed as the inventor of science in general, and especially of the healing art. The Ebers papyrus (Leipzig), is the oldest book on medicine now existing, with the exception of the Kahûn papyrus. Though much of its contents is of far greater antiquity, it was not collected together until the XVIIIth Dynasty; several later medical papyri exist in Berlin and other European museums. We know that domestic medicine chests were used by the old Egyptians, and that of Queen Mentuhotep, XVIIth dynasty, is now at Berlin. The Ebers papyrus gives a very large pharmacopœia, more than 60 vegetable substances being noted, besides minerals and animal products. The materia medica included the following drugs, still of daily use, sulphate of copper, nitrate of potash, castor oil, opium, gentian, mustard, aloes, linseed, squills, colchicum, hyoscyamus, magnesia, lime, soda, iron and others. Castor oil has been grown in Egypt for about 4000 years, and always employed as a favourite purgative, hair restorer for women, and as a sedative liniment for some skin diseases.

Most of the medical papyri mention flour, honey, beer, olive oil, incense and salt.

The Ebers papyrus consists chiefly of 811 prescriptions with

62 short diagnostic aphorisms mostly about abdominal diseases and swellings, and there are, in addition, some magical spells.

Moses (Musa) was educated at Heliopolis, and the Pentateuch presumably tells us what was known in those days of midwives, circumcision, hygiene, leprosy and skin diseases, but the old Egyptian medicine degenerated into magic and alchemy long before it was succeeded by Greek knowledge.

It is unnecessary to point out that all the early civilizations of the world were indebted to Egypt. The earliest natural philosophy seems to have been introduced into Greece by Thales (639—544 B.C.), who was a pupil of the Egyptian priests, and Pythagoras (580—489 B.C.) seems to have learned the principles of his new medical philosophy by visiting Egypt.

Herodotus wrote with wonder of the number of physicians in Egypt, including specialists for the eyes, head, teeth and internal organs, but the most remarkable thing about Egyptian medicine of that day was its non-progressive character. Knowledge stood still until the birth of Hippocrates (Abucrat, B. C. 460), who first separated medicine from philosophy. He is said to have been the son of a midwife, and though it seems doubtful whether he ever lived in Egypt, he has been adopted by the Arabs. He had great respect for the knowledge of the Egyptians, and said: "The physician must know what his predecessors have known, if he does not wish to deceive both himself and others." He is known as the "Father of Medicine", and 42 of his clinical histories exist, besides many wise sayings. The famous oath which bears his name, is probably authentic and the latter half of it may be quoted here as it represents some of the rules that should guide the physician to-day in the practice of his profession. "I will put my master who taught me medicine on the same level with the father who gave me life. My patients shall be treated by me to the best of my power and judgement in the best manner, and I will refrain myself from all evil and all injustice. I will not give poison to anyone if I am asked for it, nor will I make such a suggestion. I shall never recommend means to produce abortion, but will live and practise chastely and religiously.

Whatever house I am called to attend, I will aim at making the patients' good my chief aim, avoiding every wilful and cor-

rupting misdeed, and especially the seduction of women and of boys, free or slaves. Whatever I may see or hear in the course of my practice, or even outside the line of my professional duty in the lives of men that ought not to be noised abroad, I will not speak of, considering that such things should be kept secret. If I keep this oath without breaking it, may it be given unto me happily to enjoy life and the exercise of my art, ever held in honour among men. If I violate it, and forswear myself, may the opposite fate be my lot."

Anatomy was chiefly studied on animals, and physicians were necessarily ignorant of it until human dissection was allowed for the first time for a few years about B.C. 300 in Alexandria. We are indebted to Celsus for most of our knowledge of the Alexandrian school, which remained famous till A.D. 300. The anatomist Herophilos was one of the heads of this school, and is reported, when asked who is the best doctor, to have answered: "He who knows how to distinguish the possible from the impossible." He is famous as having been the earliest experimental physiologist. The books of Celsus fill up the historic gap between the Greek pioneers and Galen. Pliny says that the Egyptians examined bodies after death to ascertain the nature of the disease of which people had died, but this habit was probably discontinued when dissection was forbidden.

Greek physicians continued to travel to Egypt where they studied at Heliopolis, until the Ptolemies inaugurated the school at Alexandria for the followers of Herophilus and his rival Erasistratus, some of whom became famous lithotomists.

Galen, the Greek, studied medicine in Alexandria about A.D. 147—158, and later settled in Rome, where he continued his anatomical studies as far as possible on men and animals, but always advised his pupils to visit Alexandria, because there they would have the advantage of demonstrations on real human bones. He was the first to declare physiology to be the basis of medicine, and with him physiology became for the first time an independent science. For thirteen centuries his doctrines dominated medical teaching. In the time of Galen, Alexandria was the centre of the intellectual world, but during the decline of the Roman empire interest in science decayed more and more, and medicine again degenerated into magic, astrology

and alchemy, which was nothing but a series of efforts to make gold.

A demotic papyrus, written in the third century A.D., is almost entirely devoted to incantations and invocations, generally with an erotic object in view, but some paragraphs deal with the treatment of diseases of the eye, gout, and sprained foot.

Paul of Ægina, the surgeon and obstetrician, was a pupil in Alexandria, and during the whole of the Byzantine empire the best physicians were educated wholly or partly at that school.

The Alexandrian school was finally extinguished in A.D. 640, when the city was captured by the Kaliph Omar.

But in A.D. 500 human dissection had long been disallowed, the school had decayed, and the Nestorian Christians who had migrated from Alexandria to Syria were forced to settle in Persia, where their medical teaching spread. Here was taught Harit ibn Kaldah who, though a Christian, was highly thought of by the Prophet Muhammad and the first Kaliph, Abu Bekr.

It was about this time (A.D. 597) that civilization and medical learning were introduced from Rome into Saxon England by Christian monks.

A large book of Coptic medicine, dating from the 8th. century, containing 201 prescriptions for diseases of the eyes, stomach, and uterus, fistulæ, scabies and other skin diseases, can be seen at the library of the French Institute of Archæology in Cairo.

For 500 years (750—1250 A.D.) Arabic writers now represented the highest form of medicine, and the literary Arabic language was formed and spread over nearly half the then known world.

Muhammad ibn Ishak, the author of the Fihrist, says that the first translations from a foreign tongue, which were made under the rule of Islam, were works on medicine, alchemy and astronomy, translated from Greek into Arabic by the order of the Prince Khalid ibn Jazid who had been taught medicine by Marianus, a Christian monk, probably once a teacher in the Medical School of Alexandria.

The Abbaside rulers at Bagdad, especially Haroun el Rashid, and his son El Mamun, caused the prolific works of Galen,

those of Aristotle and other classics of the day to be done into Arabic.

In the middle ages physicians were well protected by their Royal masters, and often rose to the rank of Vizier. Among those specially worthy of mention are Abu Bekr Muhammad ibn Zechariah Rhazes, who first wrote on small-pox and measles, and was called the "Experimenter;" Sinan ibn Tsabet, who introduced medical examinations for license to practise; Isaac ben Solomon, an able representative of the Jews, who were very prominent in medicine during the ninth and following centuries; Ali ibn el Abbas el Madjussy, author of the Maleki; Abu el Kassim el Zahrawi, who wrote the first independent work on surgery, which was also the first illustrated treatise on that art; Abu Ali el Hussein ibn Abdullah ibn Sina, (Avicenna) famous as a doctor, astronomer, poet, philosopher and statesman, whose tomb at Hamadan in Persia is still visited by sick people in search of health, and whose "Canon," translated into Latin, formed, for four centuries, the chief text book of European medicine; Abu Merwan Abdelmalek ibn Zohr, (Avenzoar) the greatest and most original physician of the twelfth century; his pupil Muhammad Abu el Walid ibn Ahmed ibn Roshd (Averrhoës); the Rabbi Maimonides, who left Spain for Egypt and became one of the medical staff of Yusuf Salah el Deen; Muaffaq el Deen Abu Nasr Adnan ibn el Ainy, who taught medicine and wrote books in Egypt; Ibn el Faris, who also wrote several medical books in Cairo; and Abdullah ibn Ahmed Dhia el Deen, called Ibn Baitar, the most famous botanist of the East, who was for a time chief apothecary of Egypt, and wrote the Djami el Mufridat, the Materia medica of that day, and was given the title of "master" by the Cairo Academy.

According to Makrizy, the first hospital in Egypt was established by Ibn Tulun about A.D. 875, and contained a section for lunatics; the Kaliph visited it every Friday, till a lunatic threw an apple at him, after which he came no more, though he still took an interest in the place.

Kafur's hospital was built in the year 957, and Leclerc tells us that in the tenth century there was another hospital in Old Cairo.

In 1005 El Hakim founded the "House of Wisdom" in Cairo, which was practically a university where medicine was taught

besides other sciences. And another university was started in Alexandria under the Fatimites in imitation of the famous school of that city. At the end of the twelfth century there were several doctors at the Nasry Hospital in Cairo, and in the beginning of the thirteenth century Ibn Abu Ossaibiah gave his services to the Noury Hospital; both these hospitals were founded by Salah el Deen (Saladin).

Abdul Latif, of Bagdad, taught medicine in and after A. D. 1193 in the Azhar University in Cairo, and refuted some of Galen's anatomical errors.

But the most remarkable medical foundation in Cairo was the Muristan, built by El Mansur Kalaun in 1286, for he endowed it with money for wards and out-patients, and desired that all should make use of it rich and poor, great and small, bond and free. His luxurious scheme included doctors with fixed salaries, male and female nurses, special wards for fevers, wounds, ophthalmia, diarrhœa, phthisis and obstetrics; he evidently intended his gift to be a general hospital, but its traditional reputation is chiefly that of a mad-house, and it remained, in fact, the only lunatic asylum in Cairo until about 1856. The ruins of the Muristan have been utilized by the Wakfs since 1903 as an eye dispensary. Makrizy also describes the Moaiyud hospital at Cairo, which was opened for a few years about 1420.

The Arabs kept alive the torch of medical science and passed it on their successors, burning more brightly than ever, though they made no discoveries of capital importance. This was partly due to the impossibility of dissecting, and anatomy and physiology therefore remained at the stand-point they had reached in Galen's time, while operative surgery lost ground, so that tracheotomy, extraction of cataract, and many amputations were no longer considered safe, and the knife was superseded by caustics and cauteries. They were, however, more interested in practical medicine, and they invented the profession of apothecaries, whom they called "Sandalani" from the great amount of sandal wood they employed. The higher teaching institutions of the Arabs lasted till the fourteenth century, and then decayed more or less rapidly under the dominion of the Mongolian and Turkoman tribesmen. But in the Christian lands of Europe the seeds of Arabic culture developed into the vigo-

rous intellectual growth of the medical Schools of Salerno, Montpellier, Padua and Bologna. At the last named University Mondino published in 1316 his "Anatomia", which was the first work founded on actual dissection of human subjects since the days of the anatomists of Alexandria. From the Italian schools medicine was transmitted to England and Germany, and it is interesting to note that the Western nations are to-day but restoring to Egypt the modern fruits of that knowledge which was for so many years almost a monopoly in the famous cities of Memphis, Heliopolis and Alexandria. So late as 1481, the Arabist school still led at Tübingen, where the text books numbered six Arabic to three Greek works in the newly founded University, but in Egypt everything medical dwindled and decayed until there were no hospitals or schools left.

The English language is enriched by many words derived from the Arabic, and there are now in daily use many silent witnesses of the influence which the Arabs once had upon pharmaceutical knowledge, such as: alcohol, alkali, amber, attar, camphor, chemistry, coffee, elixir, lemon, musk, myrrh, naphtha, nitre, orange, saffron, senna, syrup, tamarind, taraxacum and others.

In 1820 Muhammad Ali Pasha, under pressure from Europe, laid the foundation of a military sanitary service in Egypt, and five years afterwards, under French guidance, the first board of health was established, and means were taken for treating also the civilian sick and wounded.

During the French occupation, in 1799, Kasr el Ainy palace was turned into a military hospital, and Baron Larrey studied various diseases there and lectured to the midwives. Bonaparte also ordered a commission to prepare a plan for organizing a civil hospital for the sick of Cairo, and a hospital of 300 beds was then opened in the Ezbekia quarter. Both these hospitals were disused when the French left Egypt, Kasr el Ainy was turned into barracks, became a ruin, was rebuilt by Muhammad Ali in 1812, and then became a preparatory school till 1837, when Clot Bey succeeded in getting leave to transfer to it the hospital and medical school which he had started in 1827 at Abu Za'abel. The medical school is thus the earliest of all the government schools in Egypt, and was called into existence by the ravages of plague and cholera in Egypt between 1824 and

1840. The names of the European professors that deserve mention are: Clot Bey, who, besides vaccination, introduced into modern Egypt hospitals, schools of medicine, pharmacy and midwifery, sanitary and quarantine departments, all of which, in an improved form still exist; Pruner, Griesinger, Bilharz, and Reyer. Kasr el Ainy hospital and school after 1858 were left chiefly in the hands of Egyptian professors educated in Europe, and were not productive of any scientific work. The Board of Health, which regulated the whole of the interior of Egypt, except the army, having been separated in 1881 from the Quarantine department, which became responsible at the different seaports for preventing the introduction of human and animal diseases from abroad, proved to be so incompetent during the 1883 epidemic of cholera, that it was swept away among the earliest British reforms in 1884.

Since then a new era has begun of new hospitals all over Egypt extending into the Sudan, a Sanitary Department, famous even outside Egypt for its practical methods of stamping out epidemic diseases, a Veterinary school, a model Oriental hospital at Kasr el Ainy, and a completely new School of Medicine, which has necessitated the creation of various scientific departments, which never existed until the occupation of the country by England.

Though it is now more than twelve centuries since Egypt has been under Muhammadan sway, and even longer since Christianity was introduced, there are many evidences that the bulk of the people, who are quite uneducated, still unconsciously continue some of the beliefs of the Ancient Egyptians. They believe that the dead can feel, and should be treated with respect similar to the living, and that their dead relatives in the cemeteries must be visited on important feast days. Innumerable remedies exist to counteract the dreadful effects of the "evil eye," cornelian and charcoal are put on children's foreheads for this purpose, and a monkey or gazelle is often kept in the house as a preventive. Phallic worship is by no means forgotten, and certain statues in the Cairo museum of antiquities are regularly visited by barren women. Women desiring children will also step over the body of an executed criminal or into a basin of water that has been used to wash

his corpse, or tread on a human skull, or beg leave to visit the dissecting room, or walk between the tombs of a cemetery, or step over a bronze statue of a cat, or other deity of ancient Egypt. Fevers are cured by wearing any bone which belonged to an unbeliever or to a mummy. The lowest classes often seek no medical help until the patient is moribund, but they have great faith in written charms which usually consist of passages from the Koran for Muhammadans, and of the Psalms and Gospels for Copts, intermingled with numerical combinations, diagrams and symbols; these are worn as amulets to prevent or cure disease. Evil spirits prowl about at dusk, in the body of a cat, and can easily be transferred to a healthy person, who then gets paralysis or some other nervous disease of which he can only be cured by visiting certain mosques or Coptic convents.

The varied history of Egypt must never be forgotten by the investigator who wishes to study the modern habits and customs of the people.

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INTRODUCTION TO INFECTIOUS DISEASES

In 1903 the Académie de Médecine in Paris decided that the following list of fifteen diseases ought to be subject to compulsory notification: Typhoid fever, Typhus, Variola and varioloid, Scarlet fever, Measles, Diphtheria, Pneumonia and broncho pneumonia, Miliary fever (sweating sickness, unknown in Egypt), Cholera and choleriform diseases, Plague, Yellow fever, Dysentery, Puerperal infections (when professional secrecy of the pregnancy shall not have been demanded) Ophthalmia of new-born children, and Epidemic Cerebro-spinal Meningitis. The Académie further gave the following diseases for which notification should be optional with the consent of the family: Pulmonary tubercle, Whooping cough, Influenza, Erysipelas, Mumps, Leprosy, the Ringworms, and Purulent conjunctivitis in adults (trachoma, or granular ophthalmia).

But as it is only the more conscientious doctors and householders who interest themselves in the protection of the public by notifying cases of sickness, it will be a long time before complete ideal notification will be carried out in any country.

The following fourteen diseases since 1891 must be compulsorily notified in Egypt: Small-pox, Measles, Scarlet fever, Diphtheria and Croup, Griesinger's "Bilious Typhoid fever," [1], Typhoid fever (abdom.) Typhus (exanthem), Relapsing fever Cholera, Plague, Puerperal septicæmia, Hydrophobia, Glanders and Anthrax.

All true epidemic disease is communicable from the sick to the healthy directly or indirectly, and in Egypt, Plague and Cholera are the two most important instances, besides Typhus, Influenza and Dengue.

Endemic disease is represented by Malaria, Dysentery, Diphtheria, Tubercle, Leprosy etc., while the term sporadic may be

[1] This is now held to be a variety of relapsing fever.

applied to the occasional cases of Mediterranean and Enteric fever among natives, and Scarlet fever among Europeans.

The term pandemic is used when an epidemic disease such as Influenza, Cholera or Plague becomes spread over whole countries.

It must be remembered that infection is a general term and includes contagion, for instance, pneumonia, typhoid and cholera are all infectious, but are not usually contagious.

Infectious diseases may be said to be those which are caused by living germs, which enter the body from without and are capable of multiplying in the body. The lesions produced in the body which we so often call symptoms, are due, not to these germs, but to the action of the products of these germs, that is, we have infection first and intoxication later. The period of incubation is really the time which the germs take to manufacture the poison in the human body.

Personal pre-disposition to disease may be *natural*, as in the case of man to syphilis or diphtheria, or as in the case of guinea pigs to tubercle; sometimes there is no predisposition, like the hen who is resistant to tetanus.

Or it may be *acquired* by starvation or improper diet, fatigue, loss of blood or exposure to heat, cold or damp.

Again, it may be *hereditary* from a parent, or, independent of a parent, when it is called *congenital*. Tubercle is sometimes transmitted in this way, for tubercular guinea-pigs are found to produce 25 per cent of their offspring tubercular. Disease may be transmitted in utero by direct infection, so that the child may get syphilis from the ovary or testes, or may be infected with typhoid or relapsing fever through the placental circulation.

TYPHUS

Synonyms.—Prison fever. *Ar.* Homma Typhusia.—Nausha. It must here be explained that the Arabic word Nausha does not represent one definite disease, but is used indiscriminately for typhus, relapsing fever, pneumonia, and very bad cases of enteric or influenza. It signifies therefore any acute febrile disease in which the patient becomes delirious.—The word is said to be derived from the Coptic and means "stupor."

History.—Typhus has been described in Europe since 1505, but no pandemic has occurred since 1815, for it has gradually yielded to improved sanitation in all countries. It is still, however, sometimes present in Russia, Germany, Austria and Ireland. In 1878 I saw much of it after the Russo-Turkish war, for on the Russian side alone there were said to be 100,000 sick, of whom 50,000 died.

Probably the historical outbreaks of plague in Egypt masked typhus cases, for there is no record of epidemic typhus till 1836—7, when no less than 3000 Egyptian and Sudanese soldiers were admitted to Kasr-el-Ainy hospital out of a total of 7000 sick. Pruner says that they filled up all the wards and corridors of the hospital, and communicated the disease to many of the doctors and to two thirds of the attendants in the hospital, and that one third of the patients died, mostly between the 7th and 11th days. He ascribes this epidemic, which began in the winter and lasted till the following summer, to the following causes; the soldiers were unhappy, unwilling conscripts, who had been made to march long distances under circumstances of great cruelty, they were terribly overcrowded in barracks, and fed on diet to which they were unaccustomed.

Pruner also states that typhus was very common at this time in Egyptian factories, because the workmen were shut

up in hermetically closed rooms to prevent their running away!

Griesinger saw 63 cases of typhus in 1851—2 among the Egyptian soldiers. In 1864 and in some former years malignant typhus was reported from Khartum, and in 1876 there were a few cases in Cairo. Yet when I took over the Sanitary Department in 1884 all these previous outbreaks had been forgotten, and I was assured on all sides that the disease was never seen in Egypt. This was the more inexcusable, because I have since found out that until this time typhus was present almost every year in the students's dormitories of the Medical School. This instance is enough to show to what a depth of ignorance the Egyptian doctor of that day had descended. In August 1884 "typhoid fever" was reported from a village near Zagazig, which proved to be not typhoid, but a combination of typhus and relapsing fever. In 1886, between March and June, a severe epidemic of both typhus and relapsing occurred at the old Toura convict prison, (then containing 964 prisoners) producing a mortality of 148 in April, and 106 in May, which by sanitary measures was then stamped out.

Including two of the attendants, I saw there 32 cases, of whom 21 died. Since 1884, when I introduced statistics into the Sanitary Department, typhus has been reported from a few villages every year, generally in the spring months.

Distribution.—Unfortunately, the sanitary Department is not able to assume that its provincial officials can correctly make a differential diagnosis between the various Egyptian fevers, and this is the less to be wondered at when it is remembered that many men and most women are not seen by the medical officer during life and therefore the diagnosis has to be made from the appearance of the corpse and such history as is vouchsafed by the friends. Therefore four fevers, typhus, relapsing, enteric and "bilious fever" (probably Weil's disease) were until recently all returned under the general heading of "Fièvres typhiques". The last two are distinctly rare, and therefore the bulk of the returns under this heading must be typhus and relapsing, which often co-exist in Egyptian epidemics.

During the fifteen years 1886—1900, 17,990 deaths have been reported under this combined heading; of these 1327 were Europeans, and it is fair to assume that most of them were enteric. During the same years 19,633 cases, under the combined heading, were admitted into the various hospitals and ambulance tents in the whole country, giving a mortality of 3913. Every year the number of cases admitted into hospitals is greater, which means not that the diseases are more prevalent than they used to be, but that the medical officers of health are more energetic, and the lay officials are more willing to notify and to assist in isolating infectious cases.

There are as yet no returns from the Sudan, but it may be assumed that typhus can exist during the spring months in any part of Egypt or the Sudan where persistent overcrowding and other causes exist. In both the Egyptian and English armies during the same years typhus has been unknown, because of course the men live a sanitary life, but I remember one sporadic case which mysteriously occurred in an English officer quartered in Alexandria. Typhus is still one of the dangers of the provincial prisons, which, from lack of funds, are sometimes dangerously over-crowded. One of the last serious prison epidemics took place in Alexandria early in 1902, the infection having been brought from a temporary 'lock up' in a country-district, it caused 198 cases with 51 deaths. During 1903-4, there were 144 cases at Sohag prison, with 19 deaths, 177 cases at Beni Suef prison with 12 deaths, and 203 cases in Assiut prison with 23 deaths. In every case the prison had been dangerously overcrowded, for instance at Assiut, there is accommodation provided for 474 prisoners, yet in 1903 the average number who had to be detained was 697, and the highest number during the year rose to 812. The low mortality in these three prisons is believed to be due to the fact that during the typhus epidemic, all cases of fever were temporarily returned as typhus.

Between 1891 and 1900, the typhus deaths in Cairo averaged per year 75.3 among natives and 0.7 among Europeans, while in Alexandria the figures were 5.8 among natives and 2.7 among Europeans. The chief mortality in both cities occurred between March and August.

CAUSES.

I have never seen any case under the age of five and the greatest number of patients seem to be men between 20 and 50, though, when a village is infected, there are necessarily some cases among the women too. The hospital cases consist almost entirely of Egyptians, Berberins and, to a smaller extent, the Sudanese, but I have seen a few cases among Greeks and Syrians and one in an English servant girl.

The season of the year is, as I have said, of great importance, for instance, in 1901 the cases admitted to the Infectious hospital at Abbassia numbered 40, of whom 38 were admitted between March and August, while only two entered in the other six months between September and February. At Kasr-el-Ainy hospital I had, during 11 years, 151 cases in my wards, all between February and October, but these are now never kept in the hospital, for directly the diagnosis is made, or even suspected, they are transferred to the Infectious hospital in the desert. In former days, before any Infectious hospital existed, we were less careful about dismissing cases promptly, and as a consequence some of the male hospital orderlies caught the fever and at least one of them died from it. December and January would seem to be months when typhus is the most rare. It must not be forgotten that these are the coldest months of the year, when the villagers crowd into their huts at night with their sheep, goats and poultry, and stuff up all windows and crevices with rags. Typhus discovered in the spring months (March to May) is thus always preceded by some months of overcrowding and ill ventilation. Griesinger however saw some cases in January.

Since 1868 unsuccessful attemps to penetrate the bacteriology of typhus have been in progress, but all we can now say is that there must be a specific microbe, and that it will, in all probability, be found in the blood. Until the micro-organism shall be discovered, we can only deal with the predisposing causes, which experience has taught us are favourable to the spread of the disease, and which some day will be found to be conditions which are most suitable to the microbe. It is often impossible to trace the infection, but the old doctrine of

spontaneous generation of typhus is opposed to all analogy, and need not be discussed.

The prevalence of typhus is favoured by previous illnesses, bodily fatigue and pain, loss of sleep, alcoholism, mental anxiety and depressing emotions. The chief predisposing causes of this fever can be best studied, however, by giving as a concrete case the Toura epidemic, which is so instructive that it should never be forgotten by prison administrators in Oriental countries. They are: (1) Overcrowding, (2) Deficient ventilation, (3) Uncleanliness, (4) Faulty conservancy arrangements, (5) Insufficient diet for the work required.

On March 31st, 1886, some of the convicts working at the quarries attempted to escape, two of them were shot by the sentries, others were wounded, and the ringleaders were punished on the following day; this occurrence no doubt had a distinctly depressing effect on the general body of convicts, and prepared them for the virulent epidemic which broke out early in April.

(1) Overcrowding was well exemplified in the dormitories in which the convicts were confined for twelve hours out of the twenty-four. I carefully measured the prison cells, and found that the average official space was 23 superficial and 301 cubic feet for each prisoner. This was based upon the assumption that the cells would only be occupied by 648 men, whereas, for at least six months, before the epidemic began, the convicts numbered over 900, reaching their maximum figure (1024) in March. Both the superficial and cubic space were thus reduced for some months to a dangerously low figure. In consequence of this epidemic, a commission was appointed to inspect the gaols of Lower Egypt, and it was ordained that every prisoner must have in future 40 superficial and 600 cubic feet.

(2) Only one of the four rows of cells had outside windows, the other three rows got light and ventilation from passages which had no roof openings. The cells were surrounded by the high boundary walls of the prison, which kept out the exhilarating air of the desert. The cells and corridors were whitewashed and apparently clean when empty, but a disgusting human odour prevailed everywhere, especially in the corners. There were no inlets for fresh air in the cells, the one door shut very tightly, and the windows and roof openings were far

too small for satisfactory outlets. The air was little changed by day and became extremely vitiated at night, the cells being occupied from about 6 p. m. to 6 a. m.

Mr. H. Milton, who was then superintending the stamping out of this epidemic, and I, took the opportunity of examining one of the cells at 5 a. m. so as to see it at its worst. A horrible stench greeted us as we advanced within ten yards of the entrance door to the corridors, and in one of the cells where we found eighteen convicts, the smell was so great and the heat seemed so oppressive (though only 81° F.) that we could not remain there for more than ten minutes without feeling faint. In the middle of the cell was a pail perfectly full of solid fæces and urine, without earth or disinfectants, and there was much urine on the floor near the door, lying in large puddles. The door of the cell was tightly shut, and an iron bar quite prevented exit. Many of the convicts had thickly coated tongues, and one of them complained to us of the fæcal odour from the pail, but his companions sat silently on their mats with true Oriental indifference. In the four walls of the cell I counted more than 1000 nests of bugs, and I mention this because it has been suggested that contagion may possibly be carried by these insects. We examined other cells on the same occasion but the one from which I have quoted was one of the best in the prison, because it was bounded on the north by a corridor from which we had already removed the roofing.

(3) I have said that the cells by day looked white and clean, but there was a lack of disinfectants for the cemented floors, and the mats and blankets were rolled up in the cells all day instead of being well aired. The prisoners, both healthy and sick, were very odorous, and were mostly begrimed with dirt and covered with vermin. They were chained in couples by leg irons four feet long, with heavy links. Many of them stated that they had not washed their bodies for years, and this seemed quite possible, for no water supply existed within the prison, none was brought in for washing purposes, and the convicts were never marched down to the Nile until we suggested it. We started a bath room, which was a great success, the sick men at once began to beg for baths, and lice disappeared from all the patients in less than ten days. Twenty-five wheelbarrows

full of dirt and rubbish were taken out of the prison on the first day of our reforms.

(4) I have already referred to the conservancy arrangements which consisted of a zinc pail for urine, which was obviously insufficient, as in every cell we visited in the early morning, there was urine lying on the floor. A similar pail on the floor of the cell provided drinking water for the night. During the day outdoor pails were provided on the dry earth system.

(5) The convicts in the quarries worked for eight hours a-day and had to supply 530 tons of stone every day except Friday, but those supposed to be healthy looked so ill and anæmic, with coated tongues and haggard faces, that we succeeded in getting their work reduced for a time from eight to three hours daily. On examining the diet, we found that they had no meat, a deficient amount of vegetables and fat, and an excessive quantity of bread. The quality of the bread was, therefore, all important. To the naked eye it was evident that it had been insufficiently kneaded and baked, and was adulterated with some vegetable substance, while microscopic examination showed that it contained mineral particles and an appreciable amount of straw and bran. The analyst also reported that the flour was of mixed varieties, and was deficient in nutritive gluten.

We temporarily increased the diet of the healthy by adding four ounces of rice, 16 ounces of vegetables and five ounces of meat per day.

Contagion.—Doctors and nurses practically living with the sick and saturated all day in typhus atmosphere, as was often the case in the Russo-Turkish War (1878), are extremely liable to be infected, but European colleagues in Egypt have so far escaped, perhaps because they are few in number, and do not live near the sick. I have however treated two young Egyptian doctors who were in charge of typhus outbreaks near Cairo, and I have notes of a sixth year's student who caught typhus in May 1894. There had been no case at Kasr-el-Ainy for weeks, but I found that he had been visiting a friend ill with typhus 15 days before his own symptoms began.

I have already referred to the occasional spread of the disease to hospital *termergis* (orderlies). One of them, Abdullah,

caught typhus apparently from some cases he had been nursing at Kasr-el-Ainy, in June 1894, and was transferred to the hospital tents at Abbassia. After his convalescence he returned to Kasr-el-Ainy, and caught the disease a second time in the following May, from a patient living in his own house. His second attack lasted 14 days and was as severe as the former illness for which I also treated him. This is the only instance of which I know of a second attack of this fever in Egypt.

Except in war time, typhus is not by any means so contagious as some other fevers, (influenza, dengue, measles), and though there were more than 500 sick among the Toura convicts, I calculate that only nine per cent of them contracted typhus.

Moreover none of the prison officials caught typhus during this epidemic, though many of them succumbed to relapsing fever.

Varieties.—The most common variety met with is the adynamic, with great muscular and cardiac prostration, involuntary evacuations and a tendency to collapse. In better class patients I have met with the nervous type ("brain fever") in which the rash is dark, copious and petechial and nervous symptoms predominate. In a medical student, the temperature fell on the evening of the 16th day, but five days later went up again above 40° C. (104° F.) on account of double parotid buboes which had to be lanced. In his case delirium and mental weakness continued until he left the hospital on the 38th day.

Symptoms and Course.—I have seen the eruption appear as early as the third day but more usually it comes on the fifth or sixth. If by the end of the seventh day there is no dusky red mottling on the face or trunk of a light coloured patient, the diagnosis of typhus cannot be maintained. I have seen the typical mottling, maculæ and petechiæ on skins of all colours from the white Circassian, Greek or Syrian to the light brown Nubian. Junior students seldom discover the rash for themselves when it first appears, partly because they do not search carefully enough all over the body, and partly because they are misled by the bites of mosquitoes and fleas. No eruption of any kind can be seen in negroes or dark brown men, and therefore it is of the highest importance to be able

to diagnose typhus without this symptom. The signs upon which I am accustomed to rely are a history of a few days fever, epistaxis, injected conjunctivæ, slightly contracted pupils, a dry tongue, a slight musty odour from the mouth, a feeble first sound of the heart, and a general air of prostration of mind and body. In severe cases the rash when it can be seen becomes more and more marked, and in fatal ones the petechiæ seem to take on early post mortem staining so that they are often more apparent after death than during life.

The *temperature* behaves as in Europe; in several cases the fever rises to 40.5° C. (105° F.) or above it during the first week, while a temperature of 41° in the first week is a bad sign. In favourable cases the temperature falls on the 14th day, and then remains below normal for about ten days. Occasionally the temperature falls on any other day between the 10th and 16th. As a matter of prognosis it may be remembered that a patient who is still alive on the 15th day is not likely to die of typhus though he may of course die of some complication later. Perspiration during the fever is usually only present in moribund patients, and in a few of them I have noticed that the sweat leaves a white crystalline efflorescence upon the eyelids and face. Deafness is an early symptom, and the patient, who from the beginning seems dull and stupid, gradually lies more and more like a log in bed, until in the second week coma, low muttering delirium and retention of urine appear. The delirium is seldom busy or maniacal, though the patient sometimes becomes excited when shouted at, and may even get out of bed without apparent reason. At the end of the first week when the mental condition is already blunted, a man hesitates to protrude his tongue, on invitation, and, later on, forgets to retract it until he is reminded, but in the comatose state the tongue lies in the floor of his mouth curled up, and it is only by seeing occasional tremors pass through it that you know the patient is trying to obey the command to put it out. Sometimes he is able to advance it as far as his teeth. The tongue is always dry from the beginning, and gradually becomes more dry and more brown, until it is crusted; in dying patients there is a moist, foul coating outside a black, stinking crust. The feeling of thirst seems overcome by stupor, but all except

the quite comatose, drink greedily of any liquid offered to them. Vomiting and hæmatemesis I have only seen in one patient, on the 21st day. (Case 1).

The characteristic *odour* can be lessened by giving each patient plenty of cubic space, or, what is better still, treating him in the open air. The smell is worth studying, for I have often been able to diagnose doubtful cases by it. It has been compared with many offensive odours, but it seems to me to be most like a cupboard full of well-blacked boots. During the Toura epidemic we were obliged to expose our clothes and our clinical notes to the sun for many hours before we could get rid of the smell.

Hypostatic congestion is such a common occurrence that it is rather a symptom than a complication, and is therefore useful for diagnosis in the Sudanese. It begins towards the end of the first week, and becomes more apparent till the end of the illness and is usually without cough and without expectoration. Although at least three-fourths of the cases have contracted pupils, my experience is, that the "pin-hole pupil" of the books is rare.

I have never seen or heard of any true relapse during typhus in Egypt. The *mortality* varies with the conditions under which the patients are treated, for instance, the convicts at Toura were dying at the rate of 80 per cent, but the mortality fell to 50 per cent, which is, however, enormously high, directly we supplied the sick with unlimited cubic space. There is no acute disease which lends itself more easily to open-air treatment.

Complications.—True pneumonia is very rare, and I have never seen gangrene of the lung, but hypostatic congestion, as I have said, is present more or less in every case, and bronchitis may occur in about 10 per cent. Bed sores, as in other diseases depend chiefly on the quality of the nursing, but they are extremely prone to occur during this fever on the sacrum or hip, yet I have seen some satisfactory cures in spite of enormous bed-sores occurring before admission to hospital. Buboes are most common in the parotid gland, and occasionally occur in the submaxillary, they are formed with great rapidity and ushered in by considerable tension, pain and tenderness, so that the

patient is unable to open the mouth or protrude the tongue; they may come at any time after the tenth day. Jaundice is extremely rare, but I have seen two cases which died respectively on the seventh and eighth days. Among the complications which I have never seen are: gangrene of the extremities,[1] suppuration of joints, erysipelas, and phlebitis. Several patients have been brought to the hospital suffering from mania during convalescence, and I have seen a good many convalescents who went through the disease safely at their homes, and had to be admitted for debility later. Pregnant women miscarry and often die.

Although constipation is one of the symptoms of the febrile period, diarrhœa and dysentery often appear when the patient is thought to be convalescent, so that it is important to protect him from chills and to regulate his diet to a certain extent. The only concurrent disease of great importance is relapsing fever, which sometimes comes on during a mixed epidemic after the attack of typhus. The following abridged notes will illustrate these rarer sequelæ.

Case I. *Typhus; hæmatemesis, death from dysentery on the 27th day.*—A fair Egyptian carpenter, aged 30, was first seen on the seventh day, when he had well marked eruption on his belly, chest and arms, tongue very dry and red, conjunctivæ injected, and bowels constipated. Temperature only 38°, but P. 120, and R. 30 with slight cough. *10th day*, T. 39°.2 and has not been higher. P. 132. R. 24. eruption fading, tongue dry and brown but clean. *14th day*. Temp. has fallen to 37°·5. P. 120. eruption very faint, says he is quite well, and is very anxious to get out of the typhus ward, where all his neighbours are comatose. *19th day*, feels strong, and asks to be shaved. *21st day*, had diarrhœa yesterday, which is now dysenteric, he vomits blood, tongue rough, very dry, and red brown. T. 40°·1. P. 132, quite prostrate. *24th day*, T. 37°·5, patient sensible, he passes unconsciously many motions, brown, liquid, bloody with mucus. P. 144, much emaciated. *27th day*, died at noon. I examined the body two hours afterwards, and found none of

[1] Dr. Kirton tells me that two cases occurred, in the prison hospitals, of gangrene of the lower extremities, one of which required amputation.

the ordinary signs of typhus. The blood was clotted normally, the heart looked healthy, and contained firm clot, there were hypostatic congestion of lungs and congested liver, but spleen, kidneys and small intestines were normal. Large intestine thickened and contracted, especially the rectum, with much recent congestion, and great prominence of the solitary glands of descending colon and rectum, but no ulcers.

Case 2. *Typhus, death from dysentery on 38th day.*—A dark brown Egyptian, æt. 35, was admitted on the fifth day. On the eleventh day the temp. reached its maximum of 39°·4. "P. 120, tongue dry and brown, stupor since yesterday." *14th day*, temp. fell yesterday to 36°·6. P. 102, tongue dry and red, stupor continues, minute black petechiæ on trunk. *21st day*, temp. now normal after having been sub-normal for a week, tongue clean and moist, has quite regained consciousness, feels hungry. *29th day*, dysenteric diarrhœa first began, with 20 motions, tenesmus, mucus and blood. The dysentery continued unchecked till death on the 38th day, in spite of careful diet, ipecacuanha, bismuth, opium, sulphate of copper, sulphuric acid, simaruba etc. (This patient was under my care before I knew the value of large enemata in dysentery). *Post Mortem* six hours after death. Much emaciation, no bedsores, muscles red and healthy, no ecchymoses, blood clotted but rather dark in colour, lower halves of lungs œdematous, with slight hypostatic congestion, upper halves partly collapsed and airless. Liver and kidneys congested, heart and spleen normal, mesenteric glands much enlarged, pink congestion everywhere in small intestine, but no ulcers. Solitary glands of great intestine very prominent, with many small dysenteric ulcers, white patches all through the rectum, and one large ecchymosis.

Case 3. *Typhus, dysentery on 36th day, recovery.*—A white Turkish carpenter, aet 40, was admitted on the sixth day, with temp. 39°. *7th day*, T. 38°·5. P. 120. Conjunctivæ injected, says he has pain everywhere, tongue very dry, constipation for last three days, very typical eruption on axilla fronts, belly and back, lungs resonant, slight cough, liver and spleen not enlarged, nor tender. *11th day*, eruption still clearly seen, mind stupid since yesterday. *15th day*, T. 36°, eruption fading, stupid, pupils

small, bedsore beginning on sacrum. *25th day*, sleepless at night, otherwise well, has had some boils on the forehead. *33rd day*, quite well yesterday, but says that the sun in the garden has affected him to-day (June) T. 39°·7, P. 150, tongue clean and moist, no diarrhœa, no pain, slightly giddy, sleeps well. *34th day*, T. 39°·6 a.m. P. 150, tongue very dry, thirst and headache (Sun-fever?). *36th day*, T. 36°·5, P. 126, eight dysenteric motions. *39th day*, T. 37°, has had twenty-five stools. The dysentery then gradually improved, and he left the hospital on the 64th day.

Case 4. *Typhus, parotitis, relapsing fever, recovery.*—A light brown mason was brought to the hospital on the fifth day of his illness. T. 38°. 2 p.m. P. 90. No eruption, headache, tongue dry, furred. For the next five days his fever varied from 39°·5 to 40°, and he grew steadily worse, but remained conscious. *11th day*. T. 40°, P. 126, headache, groaning with delirium, and when roused with difficulty he says he has pain all over him, tongue dry, red and fissured, conjunctivæ injected, eruption well seen on chest. Forgets to shut his mouth after opening it, spleen and liver distinctly felt below ribs. *13th day*. T. 39° P. 120, worse than yesterday, inflammation of parotid gland, but no pus in it. *20th day*. T. normal for five days, P. 84, says he is quite well and seems so. *25th day*, asks leave to move his bed away from a neighbouring patient who is dying of typhus and smells horribly. *26th day*, T. 40°, P. 108, R. 30, thirst, tongue moist; has had a rigor, and says he caught cold this morning from sleeping in a draught from the window. *27th day*, T. 38°·5 P. 138, tongue moist, red and rough, no cough, no pneumonia. *28th day*, T. 39°. 3 a.m., 39°. 7 p.m., tongue clean, no pain anywhere, but his *blood contains very distinct spirochetæ*. Transferred to another ward, containing relapsing fever patients. *30th day*, T. 40°. 2 a.m., P. 120, tongue very large, red and rough, no pains. *33rd day*, T. 38°·4, sweating much, tongue better. After the 35th day T. remained normal, till he was discharged cured on the fifty-eighth day. This case was specially interesting because I hesitated as to whether it was typhus or relapsing, when he was first admitted, and in fact until the typhus eruption appeared.

Case 5. *Typhus, bronchitis, relapsing fever with relapse, recovery.*—An Egyptian convict, delirious, with a typical eruption was admitted on what was said to be the fifth day. T. 40°, P. 124. *6th day*, tongue very dry, T. 40°·2, cough, white bronchitic sputa, impaired percussion resonance at both bases, but good breath-sounds everywhere. *9th day*, T. 39° a.m., P. 128, R. 40, tongue dry and glazed. *No spirochetæ in blood. 13th day*, is now conscious, and says he is doing well, eruption still present, T. 39°. *15th day*, T. 37°·5, tongue moist, much cough. *17th day*, T. 37°, much better and says he is quite well. *23rd day*, T. 37°. 3 a.m. P. 114, slight headache, but sitting up. *28th day*, T. 38°. 5 a.m. P. 138, giddy, pains everywhere especially in the head. *31st day*, T. 39°. 8 a.m. very giddy, headache worse, tongue moist and pale, *blood contains spirochetæ. 33rd day*, T. last night was 40° but this morning is only 35°·7, a typical crisis. The temperature then remained sub-normal for nine days, but on the 43rd day he had a relapse; T. 40°·6, being the highest record during the whole illness. P. 120, giddiness and headache again, tongue moist and clean, bowels normal, no cough. *47th day*, second crisis of the relapsing fever, T. fell from 39°·2 to 36°·1, and then remained sub-normal or normal until the patient was discharged on the sixty-third day.

Morbid Anatomy.—I have notes of seventeen typhus autopsies done by myself without counting others which died of dysentery during convalescence from the typhus. The bodies were examined at periods varying from three to twenty-three hours after death, the average interval being fifteen hours. But in spite of this precaution putrefaction had nearly always begun, though this was not noticeable in deaths on the same days from other diseases. Rigor mortis was absent or ill developed in all cases. *Eruption* was present on many corpses, generally in the form of petechiæ upon the dependent parts of the trunk and limbs, and here there was also much greater and earlier discolouration than in other diseases. I have generally been able while doing these typhus autopsies to compare the different structures with other corpses in the post mortem room. The *muscles* were dark red or brownish and very flabby and my notes speak of two well-marked ecchymoses stretching from the

umbilicus to the pubes in the rectus muscle. The *heart* in more than half the cases was soft, very flabby, and contained no clot, but in three cases the muscular tissue was apparently normal, and in two others the heart contained a little soft black clot. In practically every case if the patient dies at the end of the second or beginning of the third week the blood is scanty, very dark coloured and perfectly liquid, without any trace of clot. I should like to lay some stress upon this for it may help to diagnose after death a doubtful case of continued fever in a black patient.

The *bronchi* in my cases had their lining membranes coloured bright red, and were partly filled with frothy, tenacious mucus. The *lungs* were never healthy; at every autopsy there was much hypostatic congestion, so that the portions of lung most affected did not crepitate, but I have only seen two cases in which large pieces of lung sank in water. The lower halves of both lungs when cut open, were smooth, and not granular, of chocolate colour, and emitted on pressure a dark coloured serum, containing almost no air. Besides much œdema there was generally emphysema in the upper lobes, but sometimes they also were apparently collapsed and airless, so that it was difficult to understand how the individual could have breathed. In several cases I have seen well-marked petechiæ on the pleural surfaces. The *liver* was only normal in one case, and in nearly all others there was much congestion. I saw an instance of Frerich's "emphysema of the liver," in which pieces of the organ floated in water, though the autopsy took place as soon as possible after death. The external colour was a greenish brown, and on cutting it open, the lobules were seen to be very distinct, and the liver substance was spongy and filled with air, crepitating on pressure like a lung, and emitting a little œdematous fluid full of air bubbles.

The *spleen* was normal in one third of my cases, and congested and double its weight in most others.

The *kidneys* were congested deeply in three fourths of the bodies, and one of them had a large ecchymosis.

The *brain membranes* showed slight congestion, and were fairly adherent, though they could be stripped off without removing brain substance. Fluid in the lateral ventricles was transparent and colourless, and never seemed to exceed half a

drachm. Upon cutting the white brain substance, an unusual number of bloody points was nearly always seen.

Diagnosis.—I have never seen any case of typhus which ought to have been confounded with enteric, nor one of enteric which could be mistaken for typhus by anyone conversant with the two diseases, but I may just mention that enteric is distinctly rare among Egyptians, though, when it does occur, the student must not expect to necessarily find diarrhœa as one of the prominent symptoms. Measles presents a characteristic history of catarrh, and its victims are usually children who enjoy a comparative immunity from typhus. Pernicious malarial fever could easily be mistaken for typhus in the Sudan; it must be recognized by hæmatozoa in the blood, by the absence of history of over-crowding, by former attacks, by the season of the year and by the specific action of quinine. The three diseases from which it may be difficult to diagnose typhus are: pneumonia, relapsing fever and plague. Pneumonia in a negro will not present injected conjunctivæ, and will have earlier and more definite physical signs in the chest than in typhus, and the respiration to pulse ratio will also be higher in pneumonia.

Typical cases of relapsing and typhus are widely different, but protracted and severe cases of relapsing approaching coma, are very difficult to distinguish for a few days from mild cases of typhus appearing in negroes. In relapsing, the accession and the rigors are more sudden, the headache, giddiness and bone pains are more severe, high fever occurs earlier, eruption is absent, jaundice, vomiting and tenderness of the upper abdomen are more likely to be present, the pupils are less contracted and the odour is distinctly less offensive. In ordinary cases the moist, comparatively clean tongue of relapsing is sufficient to dispel doubt, but I have seen patients brought semi-comatose to the hospital with a dry, brown, cracked tongue very suggestive of typhus, yet even these tongues can be protruded at will, in relapsing, instead of being curled up in the floor of the mouth as in typhus. The crisis, sweating and sub-normal defervescence about the seventh day complete the diagnosis of relapsing, even in cases where the blood cannot be

examined for the spirillum. In ordinary bubonic plague, the bubo is much more likely to be in the inguinal or femoral region than in the parotid gland, it is one of the first symptoms noted, whereas typhus buboes never occur till the second week, and bacteriological examination will show the existence of the plague bacillus.

Pneumonic plague does not usually resemble typhus, but in cases of doubt the plague bacillus will be found in the sputa.

Prognosis.—This depends greatly on the age, for under twenty years only 10 per cent are likely to die, whereas after 40 years the prognosis is never good. I have already shown that in unsatisfactory conditions the death rate may mount up as high as 80 per cent. When death takes place it results from heart failure or from nervous symptoms deepening into coma, or from some complication.

Treatment.—Too much stress cannot be laid upon the importance of unlimited fresh air; in country districts cases can be best treated in open tents or huts, with only sufficient roof to protect from the sun by day and the damp by night. The patient should be allowed as many blankets as he likes and should be given unlimited drinks of water, which he always desires, though he is sometimes too delirious to ask for them.

In addition to a plentiful supply of milk and beef-tea, all patients above the age of thirty, and dangerously ill cases under that age, will be the better for stimulants, such as, brandy, carbonate of ammonium, and strong coffee. Headache requires an ice-bag and aperients, while sponging the body, and the catheter, if there is retention of urine, must not be forgotten. Buboes require to be opened early, and sleeplessness or maniacal delirium will want treating with bromide of potassium and chloral. So far as is possible, the lips, tongue and throat should be kept clean to try and obviate the occurrence of parotid buboes. During convalescence the diet must be restricted until all risk of diarrhœa is over.

"A patient with typhus is like a ship in a storm; neither the physician nor the pilot can quell the storm, but by tact, knowledge and able assistance, they may save the ship"—(Murchison).

Prevention.—Until we know and have studied the microbic cause of this disease we must confine ourselves to removing the conditions in which typhus prospers, all of which spring from poverty, ignorance, laziness and helplessness. By waging war against uncleanliness, overcrowding, ill-ventilation, and the necessary effluvia arising from these three, Egypt will be able to rid herself of typhus just as almost all Europe has done. In the meantime every sporadic case must be notified and isolated promptly, the hut or prison from which he came must be disinfected and his companions treated compulsorily to an abundance of fresh air.

A sheikh who conceals cases of "fever" in his house or in his hamlet is hardly fit to remain as head of the village!

All crowded gatherings of human beings such as schools, hospitals, prisons, barracks, and workmen's dwellings should be regularly inspected to see that ample cubic space and ventilation are provided.

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RELAPSING FEVER

Synonyms.—Famine fever, Spirillum fever. *Fr.* Fièvre à rechute, fièvre recurrente, typhus recurrent, typhus bilieux, typhus ictérodé, fièvre typhoide bilieuse de Griesinger.

Ar.—Homma el rugga, homma el naxy, nausha.

History.—Hippocrates described an epidemic of relapsing fever occurring with other fevers under his own observation in the Island of Thasos. Among other cases he quotes the sudden onset of the disease in two brothers, in one the initial fever lasted seven days, in the other six, the intermission in one five days, in the other six, and the relapse in both five days. But it is remarkable that relapsing fever does not seem to have attracted attention as a special entity for nearly twenty-two centuries after the time of Hippocrates.

In the year 1201 A.D. Egypt was cursed with a very low Nile and a consequent famine, described by Abdul Latif, during which many human beings were devoured by their relatives and others. The large town now called "Old Cairo" became almost deserted, and hundreds of dead were removed daily and left to rot outside the walls, while many of the towns and large villages in the Delta suffered to a proportionate extent. It is not surprising that a terrible pestilence should have broken out among the miserable survivors, and this was followed by an earthquake after which came a high Nile, and peace and prosperity were gradually restored, though Old Cairo was not repopulated. It is quite possible that the fever which accompanied this famine was partly relapsing, though this is mere conjecture.

In 1797—1800 the French doctors of Napoleon's army saw relapsing fever in Cairo, and Barbes pointed out that the jaundice though accompanied by relapses, resembled the yellow

fever of America. Larrey, under the heading of "Yellow fever complicating gun-shot wounds", describes a fever which succeeded the siege of Cairo in the spring of 1800; this was so sudden in its onset, so contagious and so fatal to the wounded, that the French soldiers believed that the bullets of the besieged Egyptians must have been poisonous; 260 wounded, out of about 600 attacked, died at this time, but the epidemic was stayed directly Cairo surrendered and the French besieging army was relieved from its privations and unhealthy surroundings.

The chief symptoms were jaundice, fever, thirst, pain in the head and right hypochondrium, epistaxis, vomiting of bile, occasional delirium and a marked crisis, sometimes followed by a relapse. When the city had fallen, the besieged garrison was found to be suffering from a similar disease, which was certainly what we now call relapsing fever.

Veit and Pruner wrote of a fever in Cairo, attended by a small mortality, which ended abruptly on the fifth or seventh day, but was liable to relapses, and sometimes lasted altogether 22 days. Since then Russegger and Hartmann have written of "bilious typhoid and famine fever" as being often epidemic in the poorest districts of Egypt and the Sudan (Berber and Dongola), but it was not until 1842 that physicians in Scotland pointed out the difference between typhus and relapsing, and to Griesinger was due the credit, in 1851 at Cairo, of first differentiating this fever from typhus, enteric, bilious remittent and yellow fever. He characterized it as an aggravation of relapsing fever, and gave it the name of "bilious typhoid", unfortunately, however, confusing it with the *typhus icterodes* of Smyrna, which we now call infectious jaundice.

He treated 132 cases, all Egyptians, at Kasr-el-Ainy hospital, and besides making 25 autopsies on these, he made 76 other post mortems upon cases which had died of this fever.

Griesinger's "bilious typhoid" was found to accompany epidemics of relapsing fever in Russia, Germany and India, and their complete identity was proved in 1879 by Moczutkowsky of Odessa, who, by vaccinating a man with vaccine taken direct from a "bilious typhoid" patient, transmitted to the former a typical attack of relapsing fever without jaundice.

In 1873 Obermeier, assistant to Virchow in Berlin, discovered

the spirilla in the blood of relapsing fever patients, and these were first found in Egypt by Engel Bey in 1884, But in spite of Griesinger's writings, his name and this disease were unfortunately quite unknown to me and others in Egypt, when, a week after my appointment to the Sanitary Service, I investigated, in February 1884, an illness, which had been returned as "typhoid," in a little mud village near Benha. The fever had lasted twelve days, and had already attacked 98 people of whom I examined 27. Their clean tongues, constipation, and healthy motions, made me certain that the epidemic was incorrectly named, but I was puzzled for a time until the interpreter showed me one case which was undergoing a second bout of fever after a feeble attempt to work in the fields. The chief symptoms were vomiting, temporal head-ache, prostration, constipation, temp. 40°.3, pulse 130, and no sign of eruption, with a history of only one death. Though I had never seen a case of relapsing fever, I fortunately remembered Dr. Murchison's graphic lectures on the subject and at once decided on the diagnosis, which was later confirmed by the presence of spirilla in the blood. These villagers all vowed they had plenty to eat, and the experience of this and later epidemics has been, that it is rather the quality than the quantity of food which is at fault in Egypt.

In April 1884 I again had the opportunity of seeing relapsing fever in an epidemic which had also been returned as "typhoid," from Basateen village, four miles south of Cairo. During the same year, there were five other small outbreaks of relapsing, occurring in different villages, numbering about 200 cases in all; one of them was the village near Zagazig with cases of typhus as well as relapsing, which I have already mentioned — (p. 16.)

Since my re-discovery of this disease in Egypt, small outbreaks have been reported to the Sanitary Department every spring sometimes with, and sometimes without typhus. During the Toura mixed epidemic, to which I have referred so often when discussing typhus, there were several hundred cases of relapsing, and I took notes of 118 of them.

Distribution.—This fever has been known in Uganda since 1899, but it is more commonly heard of in Lower Egypt than

in Upper Egypt or the Sudan, for it must be remembered that in the Delta, not only is the climate colder, thus leading to more overcrowding indoors during the winter, but also the population is much more numerous. It still sometimes exists in provincial prisons, in spite of improvement in hygiene, (8 deaths in the year 1902), but it is not known in the English or Egyptian armies. I have never seen an instance of it in a European, except the following: in May 1892 I was summoned to Suez to see an English doctor with slight diarrhœa and a continued temperature between 40° and 41°. I made out that he had been six days ill, and while I was hesitating as to the diagnosis, epistaxis and slight sweating occurred and the temperature fell to normal. It was then obvious that the case was one of relapsing, and the patient stated that he had been specially interested in a hospital case which he begged me to see, because he was uncertain of its nature; on examination the hospital patient also proved to be suffering from relapsing. The Englishman had a typical relapse from the 14th to the 18th days, and a second relapse from the 27th to the 29th days. During eleven years at Kasr-el-Ainy I have had 35 cases of relapsing fever in my wards, but in three of the years no case was admitted. This disease is rare in Alexandria though infectious jaundice is often seen there. The deaths from relapsing fever vary in Cairo from one in 1894 to 409 in 1898 and 392 in 1899, mostly between March and August.

Causes.—I have seen several cases in children, but not below the age of 5, neither have I notes of any case above 50. Most of my patients have been men at the hospital, but those who are able to study epidemics in the villages find plenty of women affected, and in two villages, which I visited, the females exceeded the male sick. The season of the year is said to be of no great importance in Europe, but my experience is that Egyptian epidemics occur, like typhus, between February and May. Griesinger, too, noticed that cases were most prevalent during February and March. Relapsing has been met with on several occasions in Europe during the life of the present generation, and the exemption which European residents have enjoyed in Egypt, is not referable to peculiarities of race but to the favourable circumstances in which they live as compared with the

poorest class. This fever does not occur in Egypt among any but the poorest natives, unless they have been exposed to attendance on the sick.

The predisposing causes of relapsing are very similar to those of its favourite companion typhus; over-crowding, deficient ventilation, uncleanliness, and insufficient diet for hard work. Starvation does not seem to be an important cause in spite of the disease being so often called "Famine fever," and relapsing did not occur among the starving refugees during the Mahdi's rule in the Sudan. Hard work however, is apparently a predisposing cause, for in the Tourah epidemic, where all the convicts were similarly treated as regards overcrowding, ventilation and dirt, 74 per cent of those working at the quarries at heavy labour caught relapsing, in spite of their extra diet, while the light labour convicts such as basket makers, carpenters, cooks and water-carriers, only furnished 26 per cent.

Contagion.—The liability of attendants on the sick and others to contract relapsing was well shown at Toura. Volunteers at the rate of one convict for ten sick were taken from the healthy prisoners to act as ward attendants, and, out of 74 of these, 39 caught relapsing after periods of time varying from two to six weeks. Also the Director of the prison, five warders, two clerks, and four of the black sentries whose duty it was to guard the out-side of the prison, were admitted as relapsing patients. But none of the laundry workers contracted the fever, and though Mr. H. Milton, two native house-physicians and I cut our fingers at autopsies of relapsing patients we were fortunate enough to escape inoculation. The fever has been transferred from men to monkeys by inoculating them with blood squeezed from bugs which had been allowed to suck patients, provided that the interval of time was not more than 48 hours. (Nuttall). Karlinski found spirilla in bugs in Bosnian houses infected by relapsing fever.

SYMPTOMS AND COURSE.

The first paroxysm is generally of very sudden onset. A rigor, headache, giddiness, nausea, pains, and a sense of fatigue come on without warning when a man is hard at work or

returning home after work. The rigor, however, is not always present. The duration of this first paroxysm averages 6.7 days, varying from two cases of three days to two others I have seen of twelve days each.

A postponement of the crisis till the twelfth evening is extremely rare, in my two cases spirilla were present and there were no complications to account for the length of the fever. Neither of them were followed by relapse.

Physiognomy.—A flushed face and injected eyes occur in most bad cases but never to the extent seen in typhus. During an epidemic, one can recognize a new case by the distressed expression caused by great pain, while pallor and a peaceful appearance show that the crisis has arrived.

In a mixed epidemic it is important to carefully examine all patients for eruption, so as to exclude the possibility of the presence of typhus among them. I have notes of only eight cases in which a rash could be found; in four of them there were a few faint, reddish spots which only lasted a few hours; in a fifth case I found some tiny ecchymoses in the left axilla but not elsewhere, and in the three remaining cases there was a real eruption, of which the following are notes of a mild attack.

Case 1.—A light coloured Egyptian hospital clerk aged 40, had been employed for a month in keeping diet sheets and other clerical work which took him daily into the relapsing wards. One evening he was suddenly taken ill with headache, fever, giddiness, general pains and bilious vomiting, but no rigor. *3rd day*, temperature 39°·4, pulse 112, complains greatly of headache, sleepless, bowels open after purge. *4th day*, pains better, slight headache, no eruption T. 38°6, P. 120. *5th day*, T. 39° P. 126, giddy, headache, sleeps well, small petechial eruption all over back, chest and shoulders, like large flea bites, but without puncta; feels much better but is frightened by seeing the eruption. *6th day*, crisis, Temp. sub-normal, says he is quite well and wants to return to his work, eruption completely disappeared from back, but still faintly present on chest and front of axillæ. *7th day*, Temp. sub-normal, eruption quite gone. *19th day*, convalescent without relapse, and allowed to leave the hospital.

The Temperature often reaches a maximum of 41°·1 (106° F.) though in many cases it never rises above 40°. The highest temperature which I have ever taken myself was 42°·1 (107°·9 F.). The greatest drop in temperature during the crisis that I have seen was 5° C. from 40°·4 to 35°·4. In other cases I have seen the temperature fall from 41°·1 to 36°·6 and from 40°·6 to 36°. The sudden loss of fever and subsequent sub-normal temperature for a few hours, or even a few days, is quite pathognomonic of a typical case. A very high temperature before the crisis seems to cause the patient no greater inconvenience than a moderate degree of fever.

Sweating to a slight extent takes place during the fever, and the very profuse sweat which accompanies the crisis and soaks the bed clothes for some hours is most marked. When I have examined the sweat, I have always found it to have an acid reaction. The skin remains cold and clammy for two or three days after the crisis.

The *odour* from the skin is musty, and most marked in some cases, but never so strong and penetrating as in typhus.

The *pulse* during high fever varies generally from 120 to 132, in exceptional cases it rises to 150 and once I counted it 168. In many cases the pulse is as high on the first day of the fever as later. At the crisis the pulse usually falls from about 120 to 96, but in exceptional cases I have seen it fall in a few hours from 125 to 66, or from 152 to 84.

The *respirations* usually rise and fall with the pulse rate, but I have sometimes counted them between 48 and 60 during high fever. A slight cough with scanty bronchitic sputa is occasionally present.

The *tongue* in a very large number of cases continues moist and clean throughout the attack, thereby furnishing a very useful aid in diagnosis from typhus or enteric fever, but giving no information as to the height of the fever, or as to the presence of diarrhœa. A clear, triangular space at the tip is often present, the rest of the organ being covered with a thin, whitish fur. The tongue is very seldom flabby, or indented by the teeth, is not tremulous and can nearly always be protruded. In very bad cases it may become dry, brown and fissured. During the fever, there is of course a loss of appetite, and I have never seen any voracity after the crisis.

Thirst is a constant symptom, and men though racked with pain will leave their beds to gulp down many cupfuls of water.

Vomiting occurs often at the beginning of the attack, and nausea is always complained of. The vomited matter is watery and tinged with yellow-green bile, but is never coloured by blood. Gurgling, iliac tenderness, and tympanites are not present. *Epigastric pain* is complained of by most patients at the beginning of the fever, and is sometimes very severe. During the fever there is always tenderness upon pressure in the epigastrium, or in one hypochondrium or both, though the liver usually cannot be felt to be enlarged.

The liver and spleen are, however, sometimes both painful and enlarged. One patient, on admission, complained of giddiness, pain in his spleen and epigastrium, which produced very laboured breathing, though his respiratory organs were normal; his spleen measured 7 inches vertically, and was very tender to the touch. The microscope showed spirilla, and very numerous leucocytes over 100 being in the field at once. T. 40° 1, P. 144. Two days after admission his crisis came, and the spleen slowly resumed its normal size.

Case 2.—A prison warder exposed to the infection of relapsing fever, caught that disease on May 15, and was treated for it at Kasr-el-Ainy until June 2, when he went to his own house cured. He returned to his prison and caught relapsing a second time on June 17 of the same year, when I again saw him. He was groaning with pain, which was chiefly in his back and abdomen, jaundiced, with small pupils. T. 40°·2, P. 116, spleen measured 8 inches vertically of which three inches were below the ribs, liver measured 10 inches in the right nipple line, five inches being below the ribs. *9th day*, crisis, no pain, T. 35°·5, P. 84, tongue very red, moist and rough, *10th day*, T. 36°·5, P. 72, jaundice continues, spleen measures 7 inches, the liver only six inches. *14th day*, Temp. still sub-normal, tongue clean and moist, no jaundice, spleen only measures three inches, and cannot be felt below the ribs, liver measures five inches of which half an inch below ribs. There was no relapse.

Jaundice in my experience, is present in one in ten cases, but

whereas the proportion among survivors is only 4 per cent, that among fatal cases is as high as 47 per cent. There is no doubt that this symptom is more frequent and more intense in severe than in mild cases, and I have never seen it during a relapse. When it occurs in prostrate patients with delirium and dry-brown fissured tongue, it is extremely useful in diagnosing the disease from typhus.

Constipation is present in most cases on admission.

Diarrhœa is sometimes an early symptom, and often occurs at or after the crisis. Dysentery I have seen four times in convalescing patients.

The urine is usually normal, unless patients are approaching the "typhoid state" when a little albumen and scanty secretion of urea will be found. Bile pigment is present if there is jaundice.

Headache is almost invariably frontal, it lasts all through the paroxysm, disappears at the crisis and returns again with the relapse.

Giddiness is also a very prominent feature, and is one of the earliest symptoms lasting till convalescence. Many patients fall to the ground while trying to overcome their vertigo by walking.

Bone-pains are usually considered the principal symptom by the sufferers, they begin early and usually subside with the paroxysm, but in some bad cases they continue throughout convalescence. The knees are the favourite part affected, but very often the pains are too general to be at all localized; they are never attended by local heat, redness or swelling. This is the symptom for which patients demand relief, and which afterwards remains longest in their recollection. They usually state the pains are in their bones, but this term includes also pain in the joints and neighbouring muscles.

Delirium occurs in 13 per cent, mostly during the paroxysm, but in rare cases continues for a day or two after the crisis. Occasionally the delirium is acute, noisy and shouting, but more often the patient merely seems to be unduly happy, and is often a great source of amusement to his neighbours. On one occasion a fellah staggered outside the hospital tent to drive an imaginary buffalo out of a corn-field.

Sometimes I have seen *stupor*, which lasted through the febrile

period, and well into the intermission, making it difficult to diagnose from typhus.

Sleeplessness is a common symptom due to high fever, and bone pains.

Organs of special sense.—The pupils are usually normal, but I have seen them contracted in a few cases, and dilated in others. The patients often complain of singing in the ears. Epistaxis is a rare accompaniment of the crisis, and in one of my cases was very profuse. Hyperæsthesia I have only seen once in a bad case.

Clinical stages.—After the primary paroxysm of fever come the *crisis* with its grateful relief, deep sleep, coldness of skin, and the sweating that I have already mentioned.

The *intermission* varies from six to twelve days, and may extend to fifteen. I have seen dozens of patients grumbling at being kept in hospital because they felt perfectly well, and wanted to return to their homes, yet on the following day they were again feverish and groaning with pain and thirst.

The *relapse* is by no means a certainty, but it occurred in 68 per cent of my patients, its average duration being three days, but I have several times seen it last only one day, and on one occasion it extended to nine days. At the beginning of an epidemic relapses are the rule, but as it approaches its end, they become less frequent. The relapse when it occurs, is a modified condition of the first paroxysm, and like it, comes on without warning, but in it diarrhœa seems a more frequent and more serious complication, partly because the patient is exhausted by the previous fever. I once, however, saw the temperature in the relapse, much higher than in the primary fever. A man whose maximum had been previously only 39°, reached 41° on the first day of his relapse, his temperature on the second day was 40°·9, and on the fourth day it fell to 35°·7, with the occurrence of the second crisis.

The *second crisis* is similar to the first, except that diarrhœa often attends the copious sweating and other signs of relief.

Convalescence is sometimes slow after all disappearance of the fever, but this depends chiefly on the condition of the individual before he contracted the disease. A *second relapse* occurred in

11 per cent of my cases, and never lasted more than three days, seldom being of more than one day's duration. It caused pain, and distress to the patient, but otherwise was of no clinical importance. A *third relapse* I have only seen in one case, and that only lasted one day.

No relapse took place in 25 per cent, but all these occurred at the end of epidemics.

It would seem that one attack of relapsing confers but little immunity, for besides case 2, I have seen four others who underwent a second attack of Relapsing, with generally an interval of only 4 or 5 weeks between the last relapse of the first attack and the primary paroxysm of the second attack.

A hospital attendant at Kasr-el-Ainy caught relapsing fever there in July 1893, and had it there again in May 1894.

Mortality.—Murchison's death-rate of 4 per cent is quoted in most English books, but there are records in other countries of much higher mortality. For instance, 18 per cent in India and 14·9 per cent at St. Petersburg (1865).

In Egyptian villages there are sometimes 12 cases without a single death, 11 cases with 1 death, 15 cases with 1 death, and once 33 cases towards the end of an epidemic with no death at all. On the other hand there have been larger outbreaks in villages with a mortality of 11 or 12 per cent, even when typhus has apparently been quite absent. At Toura, including all deaths, I had 17 fatal cases, or 14.4 per cent, but only 4 out of the 17 died during the first 15 days of the illness, thus reducing the rate to 3·4 per cent. One man died of peritonitis, two of pneumonia, two of dysentery, and the others from prolonged diarrhœa and exhaustion. Only one man died during the primary paroxysm, on the 6th day.

Case 3. Relapsing—Death—A light coloured convict was first seen by me on the fourth day, when his temperature was 38°·8.—Very weak, bowels open eight times after a purge, tongue moist, coated yellow, jaundice, blood showed spirilla, face purple. *5th day* T. 36°·5 P. 72—Semi-comatose, in a "typhoid state", very weak, jaundice, liver and spleen tender and large, tongue dry and coated, slight cough, but lungs resonant, except at bases behind.

6th day, T. 38°· dying. P. 108, very feeble, could hardly be counted. R. 36, very laboured, much cough, mucus in trachea, lungs quite resonant in front, tongue dry, glazed and red. Abdomen tender everywhere, jaundice still very deep, odour of breath and skin almost as bad as in typhus. No eruption except some tiny dark petechiæ on chest. *Post-mortem*—(24 hours later). Body emaciated, right lung recent red congestion. Left lung very congested, purple and œdematous, felt solid, but floated well. Liver large, yellow-brown and soft. Spleen double the normal size, bright red and pulpy. Kidneys showed recent congestion. Intestines fairly normal. Blood normal in appearance, not liquid, not dark coloured.

Four men died during the first intermission on the tenth, eleventh and fifteenth days, and the others on various days between the twenty-eighth and forty-eighth. In an unknown epidemic, it should be assumed I think, that if the death rate from fever keeps up above 5 or 6 per cent, typhus is probably present.

Complications and sequelæ.—Jaundice, diarrhœa and dysentery I have already mentioned. Bronchitis and true pneumonia are sometimes seen. Epistaxis is the only form of hæmorrhage I have observed. Marked anæmia is very common during and after convalescence, and some of the most feeble show œdema of the feet and legs. Iritis, ulceration of the cornea, herpes of lips, and boils are among the rare complications. Parotid buboes I have only seen in two cases, one of which died, they appeared during the second intermission after the relapse; very little pus escaped when they were incised, but there was great tendency to burrow towards the hyoid bone and elsewhere, and in the fatal case the anterior mastoid process of the temporal bone was laid bare.

Pathology and Morbid Anatomy.—There is a great increase of leucocytes in the blood in many cases, which accounts for the anæmia during convalescence which I have mentioned. But the important need for blood examination is, of course, the presence of the spirillum, which can be found easily with a power of 500 diameters by anyone accustomed to microscope work. When blood is taken from the washed finger of a suspected case, and

no spirilla are found in two or three specimens it may be assumed either that the case is not one of relapsing fever, or that it is being searched for too late, for it must not be forgotten that the spirilla disappear entirely from the blood from 12 to 24 hours before the temperature falls at the crisis. The spirillum must be examined for in fresh blood, but will be found more easily if a stain is used, for it shows readily with gentian violet, bismark brown, or fuchsin. Günther's method is recommended for students. I have never seen any swarms or aggregated masses of spirilla, the filaments being always unattached, in motion, and never more than 4 or 5 in the field at once. The movements of the spirilla are partly rotatory, but chiefly those of lateral progression, and they vary from a slight vibratory thrill, to very rapid motion, making it difficult to keep them under the microscope; they often attach themselves, as if in sport, to the blood cells and pull them temporarily out of shape. Inoculation experiments on monkeys produce symptoms which correspond to the disease in man with an incubation of about three days and no relapse, ending in recovery; the attack does not confer immunity upon them.

The *Spirocheta Obermeieri* differs somewhat from an ordinary spirillum, it is very flexible, its ends are pointed and it is non-cultivable. Schaudinn has lately shown that an analogous parasite, the *spirocheta Ziemanni* is a phase of a trypanosome, that it has a large nucleus and a micro-nucleus or blepharoplast, neither of which is present in a bacterial spirillum, and, moreover, that it alters its shape, contracting so as to present the form of minute oval or pear shaped bodies, each provided with a nucleus. He therefore argues that, by analogy, the spirillum of relapsing fever is probably a protozoan parasite and a phase of some trypanosome.

Condensed Post mortem notes.—Discolouration of skin, but not so marked as in typhus, healthy muscles and heart and red clotted blood, congestion of kidneys and, to a less extent, of other organs. Usually no eruption, and when jaundice has been present there is a deep yellow tint of skin, bones, and sometimes intestines, while the bile ducts are perfectly pervious, and bile is found in the duodenum. The spleen is generally twice too

large, but in 4 of my 17 cases it was of normal size and weight. The liver is slightly enlarged, if the patient dies early in the attack. If there has been much diarrhœa there will be emaciation, and the intestines are congested, and often contain round worms, while the solitary glands and Peyer's patches appear normal. In one case, 16 hours after death, there was evidence of intense peritonitis, and the liver was of a yellow-brown colour, and contained air, like a sponge. The post-mortems were performed at periods varying from 3 hours to 24 hours after death.

Diagnosis.—Yellow fever is unknown in Egypt, and enteric fever has so far only been seen among Egyptians in rare sporadic instances, and never as an epidemic. The microscopical examination of the blood will establish the diagnosis beyond doubt, especially if the observer looks for malarial hæmatozoa, as well as for the spirilla.

It is chiefly by the presence or absence of spirilla that relapsing fever can be diagnosed from the infectious jaundice of Alexandria, which was so long believed to be a variety of relapsing. But the geographical distribution must not be forgotten, for the early cases of an epidemic, occurring in the neighbourhood of Cairo, are more likely to be relapsing, while similar cases, found in or near Alexandria, will probably prove to be infectious jaundice.

In relapsing, it is chiefly the spleen which is affected, while in infectious jaundice it is the liver, and there are the additional symptoms of suppression of urine and, generally, albuminuria.

Under the heading of Typhus I have given the differential diagnosis of that disease with relapsing fever. The coincidence of these two diseases may assume various forms, sometimes they occur simultaneously in sporadic cases or in epidemic outbreaks, or typhus may greatly predominate, or, as is more frequently the case, relapsing may occur as an epidemic, while typhus appears towards its end, or, finally, the typhus epidemic may precede the relapsing cases.

In far-away country villages, where no microscope can be obtained, an unknown fever can often be diagnosed correctly by remembering the critical drop in temperature which takes

place in relapsing on the 5th, 6th or 7th day. I have tried, unsuccessfully, to convey the spirillum from country cases to the town, by allowing leeches to bite patients.

Prognosis. — Jaundice is a bad symptom, as I have shown, and petechiæ on the skin are a sign of danger. The "typhoid state" is probably dependent on failure of the kidneys to act, and most cases of it die. No patient can be considered safe until he has passed 14 days without diarrhœa or dysentery after the cessation of the fever.

Treatment. — The patients should be given as much fresh air as possible, which will be found to lower the death rate. The drug treatment can be only symptomatic, salicin or salicylate of soda may be tried for pains in the limbs and headache, constipation will require sulphate of magnesia, and the tongue should be kept clean. Patients should be allowed unlimited water to drink to assuage their thirst, and also to try and ward off uræmic symptoms. Tonics are wanted during convalescence, and patients must be allowed to return slowly to solid food, and should be warned to report the presence of diarrhœa. Attendants on the sick must be protected as far as possible by giving them sleeping quarters away from their patients, by encouraging them to expose themselves very freely to aeration, and by giving them good food, and, if necessary, tonics.

The *prevention* of the disease is similar to that given under the heading of Typhus.

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ENTERIC FEVER.

Synonyms.—Typhoid fever, Gastric fever. *Germ.* Typhus abdominalis, Ileo-typhus. *Ar.* Homma typhoidia. Nausha.

History.—Enteric fever is said to have been known to Hippocrates and Galen, but it was not until the former half of the nineteenth century that it was distinguished even in Europe from typhus. Pruner, who lived in Egypt from 1834—1846, says that typhoid was then very rare among Egyptians and negroes, but he saw cases in Greeks, Armenians and Syrians, especially among children between 3 and 15 years. He also saw a few sporadic cases in natives between 8 and 14 years old He noticed that diarrhœa was seldom a symptom, but whenever he had the chance of making an autopsy, he found in the small intestines the lesions he had seen in Europe in cases of typhoid fever. Griesinger found 15 cases of "ileo-typhus" among 1087 patients at Kasr-el-Ainy, six of which were verified by post mortems. Colucci Bey has recorded that "la fièvre typhoide" broke out in the provinces of Upper Egypt in 1860, 1862—3, and was carried in June 1862 to the Suez Canal by labourers from Upper Egypt, so that the Sanitary Department in 1863 had to prevent the arrival of any more workmen from the Upper Nile. This may have been enteric fever, but I doubt it. Cerf-Mayer reported typhoid from Alexandria in 1869, and Vauvray found it at Port Said in 1873.

In 1880 Eberth of Zürich discovered the specific bacillus in the spleen and mesenteric glands of those who had died from enteric, and the typhoid bacillus is often called by his name.

In 1883 I saw enteric fever among the English soldiers in Cairo, and a few scattered cases among Egyptian soldiers, and I found that the disease was well known to our European colleagues, though many of them were surprised at the

English method of treating cases by a pure milk diet. Since then no year has passed without cases occurring among the Europeans.

DISTRIBUTION.

Enteric fever is found all over the world, and is very common in Europe, in the United States and Canada. It exists in many places in Africa and Asia, though there is apparently some immunity in tropical and sub-tropical countries. With regard to its diffusion in Egypt, it has seemed to me that we are going through the same cycle of belief which swayed medical opinion in India. In that country, about 50 years ago, the dogma passed unchallenged that India enjoyed an absolute immunity from typhoid, a few years later it was recognized that the disease occasionally occurred, and now it is universally believed that it is by no means an uncommon disease, even among the natives.

I have often heard it stated in Egypt that no adult Egyptian suffers from the disease, and the erroneous theory has been propounded that most Egyptians have suffered from this fever as children, thus procuring an acquired immunity among the adults. I think I may dispose of this theory quite shortly by stating that I have performed, or assisted at the post mortems of several hundred children under the age of five years, most of them being compulsorily brought to the hospital after death because they belonged to the foundling department of the Egyptian Government. These children were formerly farmed out to the poorest women in Cairo and Alexandria, who lived in insanitary huts, many of which contained stinking latrines. The unfortunate children died of diarrhœa, broncho-pneumonia and various forms of septicæmia, but never in one single case have we ever found pathological evidence of enteric fever, while those cases which I have carefully watched during life, to elucidate this very question, have never displayed clinical symptoms of this disease, nor answered to the serum test.

However difficult it may be to explain, it is a fact that, whereas enteric is very seldom absent from the European hospitals in Egypt, it is a very rare visitor to the wards of Egyptian Government hospitals which are frequented almost entirely by the natives

of the country. This is true of some 25 hospitals, but I need only give details of the largest of them. At Kasr-el-Ainy I saw a case in 1890 of a Berberin cook from the Barrage, who lived with an English official there; no one else in the house had the disease and his was a mild attack, but, in my opinion an undoubted one. After that I had to wait until 1898 before I could get a hospital case to show to the students. In that year two were admitted, two others in 1900, and one in 1901, thus giving in all 6 cases admitted in 12 years, out of a total of 8752 patients admitted to my half of the medical wards.

During the early part of these 12 years, I conducted the autopsies, and never met with a case of enteric in the post mortem room; when I was succeeded by Dr Kaufmann, he found one doubtful case in 1100 post mortems. Since then Dr Symmers has published a report on 835 post mortems, extending over 3 years, without one single case. These figures are enough to show either that enteric is very rare among those who attend Government hospitals, or else that those suffering from that disease do not seek relief for it at the hospital. But, in this connection, I must state that many of the hospital patients consist of policemen and other Government employés who are obliged to come to State hospitals when they are on the sick list. Since 1902 a greater proportion of enteric patients has been admitted to Kasr-el-Ainy, both Egyptians and Sudanese, and the pathological museum now possesses a sample of typical ulcers from a black patient.

The following figures show that the Egyptian troops suffer much less than the English soldiers.

	Average Strength	Average Cases of Enteric	Per 10,000
English Army in Egypt 1888—1902	4045	105	259
Egyptian Army in Egypt 1892—1902	2837	5.8	20
Egyptian Army in Sudan 1892—1902	15,323	32.6	21

It must not be forgotten too that the Egyptian Army, both in Egypt and the Sudan, consists not only of native troops, but of several British officers and a few British non-commissioned officers, who help to increase the enteric sick rate.

The peasant population of the villages is not provided with any conservancy arrangements, and therefore performs all acts of nature upon the ground in the open air, a proceeding which would be impossible except in a land of daily sunshine. The unclean habits of the villagers and their carelessness as regards food and drinking water pre-dispose them to many diseases, but not, apparently, to enteric. When a recruit leaves his village to enter the army, he is drilled, well-fed, given filtered water to drink, and consorts with other picked, healthy men, and improves consequently in general health. But he becomes liable to a slight amount of enteric fever.

Yet, when, after five years service, the soldier is transferred to the police, or returns to his village, he seems to regain his immunity from enteric. In order to try and explain this, it must not be forgotten that the soldier, during his army service is at a most susceptible age, (say 20 to 25 years) and also that while army cases of enteric cannot remain undiscovered and must be sent to hospital, where they are recorded, there is no such compulsion among the civilian fellaheen, who may, therefore, suffer more than one thinks from sporadic enteric, disguised under the name of "*nausha*."

But in spite of this, I have sometimes wondered whether the enteric fever in both English and Egyptian armies is not encouraged by the latrine methods employed; theoretically, earth and disinfectants are freely used, but practically great care can not always be taken to prevent enteric fæces and urine from becoming dried, pulverized, and swallowed with food by a susceptible individual before the typhoid bacillus is already dead.

Besides Egypt and India, a similar marked susceptibility of the European over the native is seen in the Malay Peninsula, in the Dutch East Indies, in China, and in Algeria.

The comparative immunity of the rural population in Egypt does not extend to dwellers in towns, for enteric fever is now met with in Egyptians every year in Cairo, Alexandria, Port Said, Suez and other places, chiefly during the summer. At

Suez, for instance, there is a large staff of English clerks of susceptible age. In July and August 1891 there were eight cases among them, aged 18—26, besides eight other cases among natives in the town. Since then there have not been so many patients at any one time, but only dropping cases now and then.

I am not at all alone in the belief that Egyptian town dwellers now suffer more from enteric fever than they did fifteen or twenty years ago. Though I attended a Coptic interpreter suffering from this disease in Cairo in 1883 it was quite rare for many years to be called into consultation on a case of Egyptian enteric. But for the last few years this has been by no means an uncommon experience. If the Egyptian becomes in the future as liable as the European to this fever, the Sanitary Department will have a hard task before it. I cannot quote statistics of the number of cases of enteric in Egypt every year because several fevers until 1903 were returned under only one heading, but, in an unsuccessful attempt to induce the Government to establish a drainage system for Cairo, I collected most of the cases occurring in the seven winter months of 1890—1. The four chief hospitals and the private practice of 14 leading doctors belonging to six different European nationalities gave me a record of 102 cases and of these 96 were contracted in Cairo.

Between 1891 and 1900 the average yearly deaths from enteric in Cairo were 314·3 among natives and 26·8 among Europeans, while Dr. Engel's statistics give for Alexandria 96 native and 36·2 European deaths. There is, however, no means of knowing whether the diagnosis was accurate.

CAUSES.

Age.—In the English Army of Occupation the majority of cases occur between 20 and 25, and the highest admission rate is during the soldier's first three years in the country, so that admissions from enteric are greatly reduced after the fourth year in the command. For many years in Egypt I had not seen a patient under four years or above 57 years old, but in November 1901 I had at the same time two patients under my care, one an English child aged 18 months, and the other an Egyptian

in the hospital wards, aged 65, both verified by serum tests.

In January 1905, Dr. Beddoe and Dr. Nolan treated an English hand-fed child, aged 6 months, for fever lasting 14 days, which was apparently enteric. The belly was distended and the spleen enlarged, but there were no spots and the serum test was negative.

My own hospital cases are of course not numerous enough to quote as statistics, but I have notes of 91 private patients, whose attacks I have either attended entirely or superintended. Of these 21 were under 15, only 6 between 15 and 20, 42 between 20 and 30, twelve between 30 and 40, eight between 40 and 50, and two between 50 and 57. Seven of them were under five years of age. The apparent freedom of cases between the ages of 15 and 20 is due to the fact that most Europeans of that age are absent from Egypt for educational reasons.

Of 119 fatal cases among the English soldiers in Alexandria only two were over 30, and only 18 exceeded 25 years.

Sex.—In Europe and America, both sexes are equally liable, but of my cases only 35·6 per cent were males and 64·4 females, and if we include only the patients above 20 years, the male rate falls to 31·6 per cent or less than one third. This is enough to show that the young woman, whether visitor or resident, is more susceptible in Cairo than the young man, and therefore requires greater protection.

The *Nationality* of my private patients is as follows: four Egyptians, two Greeks, one Belgian, five Americans, and 79 English, but it must be stated that the greater part of my practice has been among the English and the Americans. The two Greek cases are interesting, for they were mother and child. I attended the mother when she was five months pregnant with this child, and I thought I was justified in predicting that the child would not get enteric fever, but when she was two years old she contracted a mild, typical attack.

The patients all belonged to the professional, or well-to-do classes, 39 of them were winter visitors, and of the remaining 52 residents, six were sick-nurses, three of whom were nursing enteric cases at the time, and certainly caught the disease from their patients. My cases also include a daughter who caught it just after nursing her mother, and two mothers who became

ill while nursing their children. Five doctors are included in the series, but none of them were attending enteric fever cases at the time and there is no evidence that they caught the disease directly from patients.

One of my patients was infected in England or France, and four others contracted the poison in Italy, on their way to Egypt, while three more brought the disease with them from Palestine. Of the remaining cases, ten caught it in Upper Egypt, four in Alexandria, and two in Tanta, while for 67 Cairo and its suburbs must be held responsible. It is, unfortunately, not a very rare experience to hear of Europeans leaving Egypt apparently in perfect health and yet developing enteric on the steamer or in Europe on their way homewards.

Two thirds of my cases were either strangers to Egypt or had been in residence for less than a year.

Prevalent season.—The European statement that autumn months are the most productive of enteric is not borne out by my private cases, for I find that the winter months are even more dangerous in Cairo. Omitting decimals, the monthly percentage is: January 15, February 20, March 17, April 5, May 4, June 1, July 1, August 1, September 6, October 11, November 8. December 11. The diminution of cases in the hot months is partly due to the fact that everyone gets away at that time, especially women and children, but it should be remembered that in many tropical and sub-tropical regions the hottest months of the year are found to be the most productive of enteric. During the last 10 years the British troops in Alexandria have suffered most from September to December and least between February and April, but in Cairo, where the garrison is three times larger, the most prevalent months for enteric in the Army of Occupation are October to December and then April and May, while the least incidence is shown in February.

Contact with previous cases.—It used to be held that enteric fever could not be communicated from person to person, but, during the last ten years, dozens of cases have been reported showing that infection by contact is quite possible when there

is no scrupulous cleanliness in the management of previous cases.

The disease begins insidiously and it often happens that the patient has been sharing a bed with others or that the member of the family who is nursing the sick one, is, with unwashed hands, also preparing food for the rest of the family. Enteric is of course not directly infectious in the same degree as typhus or relapsing fever, but I am convinced that it is indirectly spread by way of infective urine and fæces to healthy persons coming in the way of personal infection. Recent army reports tell us of "comrade infection" in tents during the American campaign of 1898.

Campaigning is a very important predisposing cause in all armies, hence the French term "manœuvre fever". In spite of many sad lessons, General Officers commanding European troops in the field do not seem yet to understand that Enteric Fever is one of the most dangerous enemies they have to fight. In 1897, the enteric admissions in the British Army of Occupation during the year were 67 per 10,000 men, but in the following year, being that of the Soudan expedition, the admission rate rose to 810, though it fell again in 1899 to 75. There is nothing exceptional in this, for in the Spanish American war of 1898, one fifth of the United States soldiers, suffered from enteric, and in the two years of the South African war the English had 31,118 cases of enteric fever, with 6177 deaths. Camp pollution, flies and dust as carriers of contagion into unprotected food, and the transportation of the poison in the clothing, are the chief additional reasons why enteric has so wide-spread a prevalence in camps during a war.

Enteric in the Egyptian Army in the Sudan also reached its highest figures in 1896—7, during the advance on Khartum.

Drinking water and Ice.—A few years ago it was thought that enteric was entirely a water-borne disease, but now, very many other means of conveying the typhoid bacillus to the stomach of man have been discovered. In Egypt cholera has often contaminated the canal, pond or well from which a town or village gets its drinking supply, but there is as yet no record of a similar infection from enteric. Anyone acquainted with

the habits of the lower class Egyptian, and with the history of water-borne epidemics in other countries, can only suppose that the bacillus is not a common inhabitant of the excreta from which the country wells are so little protected.

In individual cases of the disease it is generally impossible to know by what mode the poison has entered, and patients, especially if Americans, will often state that while travelling they have never drunk any plain water nor unboiled milk. But occasionally one can trace the incubation back to a special day when an imprudence was committed. For instance: (1) An English officer in June 1894, while examining hundreds of men for a recruiting commission, and suffering agonies of thirst in the hot weather, drank some doubtful water at Tanta, and two weeks afterwards developed enteric in Cairo. (2) In May 1903 an English boy was taken by his father to climb the Pyramid, when, exhausted by heat and fatigue, he drank some water from the *goula* of an Arab; 16 days later he began a mild attack, which was verified by the serum test, yet, before and after his visit to the Pyramids he had been fenced in by all the care and precaution against typhoid which his anxious parents could devise. Tanta and the villages near the Pyramids were both believed at the times mentioned to be free from enteric, and these are only two instances among many others which tend to prove the immunity of the Egyptian to this disease to which the English and the Americans seem to be particularly predisposed. (3) An English child aged 8, whose mother had a nervous dread of the possibility of enteric, and who was therefore surrounded with more than usual care, was one hot day taken for a long ride by her father and became very thirsty; after some persuasion, she drank a tumbler of water from a well in the court-yard of the English riding master's house, because she was assured that the family, which consisted only of adults, always drank from it. Yet three weeks later she developed enteric, and no other cause for it could be discovered. This is probably an example of the relative immunity which comes by acclimatisation rather quickly to adults living in hot countries.

(4) An English regiment during a forced march in hot weather near Dongola, exhausted the drinking water, and,

maddened by thirst, rushed to an obviously unclean pond which they came upon towards the end of the day. As a direct consequence of this act, some 200 soldiers contracted enteric fever or dysentery, and 52 died.

I have never seen a case which could be traced directly to ice, though the bacilli have been known to live as long as 18 weeks in ice, the majority, however, dying within two weeks.

In October 1902, 39 cases occurred at Ogdensburg which were traced to ice which had been taken from the St. Lawrence river eight months before. The ice when melted yielded, besides other bacteria, a pure culture of the typhoid bacillus.

Milk is said to be the cause of 17 per cent of all typhoid epidemics, chiefly by means of infected water, or by a person continuing to work in a dairy while he is suffering from a mild attack. In the spring of 1899 a score of cases occurred in Cairo mostly among the children of English people who were most careful about their domestic milk supply. The cause remained a mystery until it leaked out that there had been a case of typhoid at the common dairy from which these families drew their milk. Seven of these children were under my care at the same time. It is well-known that in milk the bacilli undergo rapid development without changing the appearance of the milk and they may live for several days in butter made from infected cream. Osler says "one or two typhoid bacilli in a glass of water may be, and probably often are, taken with impunity by an individual not specially susceptible, but a few in water used to rinse a milk can or jug would find in the milk such a suitable medium for growth that in 24 hours the milk would be highly infective."

Ice-creams.—I have been able to trace one case to this cause. An English boy well protected as regards water, milk, salad etc., living in a sanitary house, used to spend his spare pennies in buying this refreshment from a man in the street. While attending this boy I became aware of others of his comrades who had apparently also contracted this fever in the same way.

Oysters are eaten from both Alexandria and Port Said fisheries, and the more careful inhabitants of these towns think they are not sufficiently protected from sewage contamination; they are believed to have caused some cases of typhoid at Port Said

since 1889. I have seen two cases in Cairo, who apparently caught enteric from eating oysters in Italy.

Fried fish.—Since 1892 six outbreaks have in England been traced to this cause, the fish not only being imperfectly cooked, but also insufficiently cleansed.

Salad and uncooked vegetables.—These are of obvious danger because the peasants on their way to market wash their green stuff on banks which are often freely polluted with human urine and fæces. The child of an Italian nobleman is believed to have got enteric fever in Cairo in 1903 from eating raw radishes; he never drank unboiled water or milk and never ate salad, but was devoted to radishes.

Flies.—One of the sayings attributed to the Prophet Muhammad is, "If a fly fall into a drinking water vessel, that vessel must be washed, for a fly carries disease in one of its wings."

Flies are scavengers to be found everywhere in Egypt and the Sudan, and often alight on milk or other exposed food when coming direct from filth. They also endeavour to crawl about the face and mouth of human beings, and are most persistent in this, evidently searching for moisture. The infective matter conveyed by flies appears to be attached not only to their heads, but also to their legs, wings and bodies. The common house mosquito is bred in cesspools and may also be able to pollute eatables.

Dust is probably often responsible for the direct infection of food, such as milk, and this is helped on by sandstorms in the spring and a considerable velocity of wind all through the year. The bacilli retain their vitality in garden earth for 21 days, in filter sand 82 days, in street dust 30 days, on linen 60 days, and on wood 32 days. (Firth and Horrocks).

An English child in Alexandria apparently contracted typhoid by dust carried from a smelling heap of recently imported rubbish which had been shot down about 300 yards to windward of the patient's house. The child, aged three, took all his meals at home, and lived on food specially cooked for him in the nursery. Other people living in the same house and near it were made ill by the smell, which gave them headaches and sore throats, but not typhoid.

My youngest case, of 18 months old, was also a well-cared-

for English child, living at Abbassia, chiefly on buffalo milk from an animal kept in the garden; the supposition in this case is that the patient contracted typhoid by dust blown from a large collection of dried sewage stored in the direction of the prevailing wind. The dust gains entrance very easily to the mouth and may be swallowed, or may primarily infect the lungs.

The *urine* is one of the chief means of spreading enteric, for though it is only dangerous in one quarter of the cases, and probably only when the patient is approaching convalescence, the typhoid bacilli when they do occur are nearly always in pure culture, so that each drop of urine may contain several million micro-organisms. A hospital nurse once accidentally drank a small quantity of urine which had been passed by a typhoid patient into a wineglass, and after 12 days she developed enteric fever.

Clothing.—Firth and Horrocks have proved that if a piece of soldier's uniform be fouled by liquid enteric fæces and then allowed to dry, the bacillus can be recovered from it 16 days later; also that from similar experiments with solid or semi solid fæces, the bacillus can be found up to the ninth day. Again, that the micro-organism is able to survive in surface soil after exposure to 122 hours of direct sunshine (in England), extending over a period of 21 days, and that a piece of infected cloth will yield the bacillus after being exposed to 50 hours of sunshine spread over a period of ten days. Laboratory experiments show that clothing, inoculated with an emulsion of the bacillus and then allowed to become quite dry, will contain the micro-organism for even eleven or twelve weeks.

Not only clothing but bedding and tents may act as agents to preserve and spread the disease, and it must not be forgotten that an undiagnosed patient is quite as dangerous to his neighbours as a recognized case.

In April 1903, ten cases of enteric occurred on a ship near London which were traced to army blankets recently brought back from the South African war; they were then found to contain innumerable typhoid bacilli. The blankets were part of a consignment of 20,000 which had already been sold at a low price to 289 persons or firms in 250 different towns in Great Britain. Warning was sent to the medical officers of health of

these towns, the blankets were seized and disinfected and no further cases of typhoid occurred.

Though water is the chief cause of outbreaks of enteric, it must be conceded that infective material carried by clothing, wind and flies may play an important part in the development of the disease.

Insanitary houses.—Untrapped drains and cesspools, ventilating directly into the house, cannot actually produce enteric fever, but they undoubtedly predispose individuals to it. For some few years after the English occupied Egypt, there was not a single house or other building which did not offend every law of hygiene. But when, by personal influence, we succeeded in introducing sanitation into a few hotels and houses in Cairo, the freedom from enteric in these caused many others to follow the good example. These four instances among many others will show the intimate connection between this fever and insanitary dwelling places, if it be remembered always that it is only the susceptible who contract the disease.

(1) Kasr-el-Ainy hospital in the earliest reforms was made less insanitary by substituting a daily removal system of fæces for open latrines in the building, but slop water and urine were still allowed to run into underground culverts which did duty as drains, for we adopted the ruling of the sanitary officers of the British army of that day, that protection from fæces was the only important essential. In 1884 an English resident medical officer was appointed for the first time, and four years later two English sisters were introduced to superintend the female wards near which they were given bedrooms. All these three Europeans developed enteric soon after arrival and one died, yet none of the Egyptian residents fell ill, and the same immunity was enjoyed by a family of English children who were the only other Europeans in residence (one of these children caught enteric a few years later when living in the town). The existing underground culverts were then all dug up and filled in, and a system of drains and cemented cesspools was established for water refuse. During the last 15 years the European resident staff (aged 25—35) has been greatly multiplied, but no fresh case of enteric occurred till the autumn of 1904 when two of the English nurses contracted it, after drinking doubtful water near the Pyramids.

(2) The Hareem hospital forms part of the Abbassia barracks occupied by the British troops. In the summer of 1890 I saw in consultation the wife of an English officer suffering from phlegmasia dolens and ulcerated sore throat after a miscarriage, which made me suspect sewage poisoning. I found that a disused latrine had been cemented over, and was used as a store room for groceries, but a small hole, carrying off water after washing the floor had been forgotten and communicated directly with a long conduit, running parallel with the quarters of several regimental messes, whose bath and sink water ran into it.

During the next 18 months six cases of typhoid occurred in regimental mess waiters and one English lady, who all lived in the adjoining quarters. As a consequence of this the conduit was filled up, the sink water was otherwise disposed of, and no more cases of enteric have occurred.

(3) In 1884 six young English police cadets were sent to live in a hut at Gezira which had been used as officers's quarters by the old Egyptian Army. One room in the hut had evidently been used as a latrine, but the Royal Engineer officer thought it sufficient to cement the floor and to provide the cadets with earth closets; all of the six caught enteric fever and were removed to hospital, but none died. When the cesspool under the hut was opened up, it was found so enormous that it took several men six days with carts to empty it: it was then filled in, and, though many English officers have since lived in the same hut, there have been no further cases of enteric fever.

(4) In 1891 the English nurse of an officer's family developed typhoid in the Bijou palace at the Citadel, and the next successors to these quarters suffered from fever, though it was not called typhoid. In 1892-3 another officer's wife quartered there suffered constantly from a mysterious fever, which did not appear to be typhoid, but in the following winter, when her husband was moved to Abbassia, she lost this fever and has had none since. In October 1893 another married officer was ordered into these quarters. His three children had already passed four years in Cairo, always in the best of health; the two elder children aged 8 and 9 had had enteric fever before coming to Egypt, but they suffered constantly from "fever"

till May 1894 when they were taken to a house in Cairo, where they at once lost all tendency to fever and improved in health, weight and strength. Their youngest sister, aged 6, had already contracted enteric in March, and was ill for six weeks, including a relapse. I then persuaded the authorities to dig up the floor in the neighbourhood of the child's nursery, though the Royal Engineers said they were sure there were no drains there, but confessed that they had no plan of the Citadel underground. When the floor was removed they found four large disused conduits, about 10 ft. deep, and 3 ft. wide, running in all directions, one of them was close to the nursery wall, and a damp patch in the wall of this conduit corresponded with a damp spot on the floor of the nursery. These quarters were then condemned, and remained unoccupied till 1896 when an army medical officer, at his own urgent request, was allowed to occupy them temporarily, after the previous history had been fully explained to him. His wife and two young children all developed enteric, and since then this old palace has been definitely abandoned.

I could tell, but it is not necessary, similar instances of enteric occurring to various generations of householders in Cairo, and similar stories with regard to army quarters in Alexandria.

It used to be believed that cesspools containing only sink and bath water were not dangerously productive of sewer gas, but in May 1894 an English corporal and an Egyptian labourer were suffocated by gas from such a cesspool at the house hired for the General by the British Government. The native accidentally dropped in, and the corporal went after him to try and rescue him, a second soldier tied a rope round his waist and tried ineffectually to save them, but was hauled up unconscious.

Swallowing a culture.—A nurse of a Paris hospital in 1903 tried to commit suicide by swallowing two tubes of pure culture of the typhoid bacillus. For two days she felt quite well, but on the third day she had headache without fever. On the 6th day she felt heavy and stupid, and was obliged to take to her bed. On the 7th day her temperature was raised for the first time, and rose on the evening of the 8th day to 40°·2 C. when

she also had rose spots and epistaxis. Serum reaction was positive on the 10th day and a very severe attack followed.

Varieties.—Of my 91 cases, 24 belonged to the mild and abortive type. Many of these, but not all, were in children, and five of them had fever for only 14 and 15 days, and were not followed by a relapse. The grave type was represented by 63 cases, and the latent or ambulatory form by three cases. One of these three was an English valet, who contracted enteric in Cairo, after which he travelled with his master to Wady Halfa and returned to Cairo on the 15th day of the disease. He had fever until the 39th day with peritonitis, hiccough and vomiting, but recovered after a long convalescence. The second was originally a mild attack, the first two weeks of which were spent by an English clergyman in travelling in Palestine and in being confined for a few days in quarantine in Alexandria, where the medical officer congratulated him on his healthy appearance; after arrival in Cairo, he had a long relapse, rigors, and dangerous hæmorrhage. The third case was that of a hospital nurse in charge of a private patient, who walked about and refused to see a doctor until the 12th day in spite of fever above 40°, she had a history of kidney disease, lung delicacy and malaria, and died on the 40th day of pneumonia. Ambulatory cases are very common among English soldiers, and I have seen several, both in Egypt and in South Africa, who did not report themselves sick until the third week of the fever, when they too often died of hæmorrhage or perforation within a few days.

The term *paratyphoid* has been recently introduced in France, Germany and America, to describe some 84 cases which have been reported since 1896, which resemble enteric fever clinically, but in which the Widal reaction is persistently absent. In these cases the diagnosis has been made by cultivating a paratyphoid bacillus from the urine or fæces and showing that it was agglutinated by the patient's blood. If this bacillus cannot be cultivated from the blood, urine, fæces or some lesion, the diagnosis is justified if the blood agglutinates a paratyphoid bacillus in high dilution, (say 1 in 200) and fails to agglutinate the typhoid bacillus or agglutinates it only in very low dilution, such as 1 in 10. There are several different strains of the typhoid bacillus,

and it seems necessary at present in those cases where the agglutination reaction is absent, to further test the blood with both the α and β strains of the paratyphoid bacillus.

Typho-malarial fever was a term introduced by the Americans about 1861, before the days when accurate diagnosis had been made possible by Widal reaction on the one hand, and microscopic evidence of hæmatozoa on the other. It is a term which should never be used, for, as a matter of fact, when the two diseases co-exist, it is merely a coincidence. In the rare cases when both are present the malaria does not usually show itself until the period of convalescence of the enteric fever, and then yields readily to quinine.

SYMPTOMS AND COURSE.

No disease varies more than enteric, and it is therefore one of the most interesting to treat. The symptoms seem to be similar all over the world, with certain small exceptions. The *onset* is always said to be very insidious, so that patients are seldom seen before the fourth day. But the earliest day of recognition of the fever depends chiefly on the intelligence of the patient, and nearly all of them can fix the first day of the illness, although they would not give in to it, or seek advice until several days later. A careful mother can generally recognize that her child is ill on the first day, from such symptoms as lassitude, want of appetite, headache, fretfulness and feverishness, but no one is justified in diagnosing the disease until the fourth or fifth day at the earliest. The onset is often masked by its similarity with some reigning epidemic, such as influenza, and in two of my cases I have been completely deceived in the real nature of the disease because they were both suddenly ushered in with pain in the abdomen, and dysenteric diarrhœa. It was not until we found that the fever remained steadily up, though the other symptoms had yielded to treatment, that enteric fever was suspected, and eventually verified by the serum test.

Enteric may always be suspected if headache is present, and if there is, even on one morning, a temperature of about 39° (102°·2 F.), with no apparent cause for the fever. Again, if the temperature reaches 40° (104° F.) on the first or second day,

we are safe in predicting that the case is not one of enteric.

The *eruption* cannot be seen on Egyptians as a rule, but is very marked on white skins, and is by no means confined to the abdomen, chest and back; but in some very mild cases in Europeans I have never succeeded in finding any spots at all.

Gurgling upon pressure in the right iliac fossa seems to me of no diagnostic value, because it depends only on the presence of air and liquid fæces in the intestine, which do not of necessity imply the presence of typhoid, and cannot be present in constipated cases. But tenderness upon slight pressure, especially if it is confined to the right iliac region, is extremely useful in doubtful cases, though here, again, it is hardly present when the patient is constipated. The less pressure that is put on an enteric abdomen the better for the patient.

Bowels.—I find that nearly half my cases, 46 per cent, had more or less diarrhœa for the greater part of the feverish period, after which they developed constipation, as is customary in this disease. Six per cent, being all mild cases, had neither constipation nor diarrhœa. The remaining 48 per cent suffered from constipation throughout the whole illness, so that enemas had to be employed every day, or every second day. This high rate of constipation is not in accordance with most text books on medicine, but lest it should be thought to be due entirely to climatic reasons, I may remind readers that during the Maidstone epidemic (1897) constipation occurred in 50 per cent of the cases, also in 34 per cent in cases treated at the Johns Hopkins hospital, and, further, the cases treated in the Yeomanry hospitals during the South African war, gave a record of 42·7 per cent.

Temperature.—Many of my cases, on several occasions, showed a temperature of 40°·5, the highest temperature reached was 41°·6 (107° F.) being on the 19th morning of an attack, which eventually ended favourably. In an adult, the lowest maximum temperature I have had was 39°·4 (103° F.), but in the child of 18 months old the temperature, taken every four hours, never reached higher than 38°·8. I have never seen an afebrile case.

The average number of days of all the cases from the beginning of the illness to the first drop of the morning temperature

to normal was 27 days, but I have already mentioned that some of the slight cases were feverish for less than the usual three weeks. I am sure that I have had more than my share of very long bouts of the primary fever, for in six of my cases the temperature never dropped once to normal for more than six weeks, and in the longest case the fever remained without a break for 55 days. All the six unusually protracted cases recovered, and only one of them had a relapse, but the disappointment in these cases to both patients and friends, who have, perhaps, made up their minds that the illness would only last three weeks, can be easily imagined.

Among minor symptoms, which are not usually recorded, it should be mentioned that all patients, after a serious attack, lose most of their hair during convalescence, and that it is quite common to see desquamation from the skin in fine branny scales, also that they suffer for a month or two from very evident anæmia.

Seven of the cases had *relapses*, never due so far as I could discover to errors of diet or nursing. The longest relapse lasted 21 days, it was that of the ambulatory clergyman. Another case had a primary attack of fever for 42 days, and then a relapse lasting from the 52nd to the 60th day.

The only case which had more than one relapse was the following: An English lady, aged 57, left England on December 30th 1895, she slept one night at Calais, and another at Marseilles, but could not remember drinking any water in France. She reached Cairo on Jan. 8th, and the enteric fever apparently began six days later. On January 19th I was asked to see her because she had fainted while dressing, and had bruised her face by falling against a marble slab. I found that her temperature was 39°, but as she had recently been exposed to the risk of influenza, I assumed it was that disease, especially as she had a weak heart, which might have accounted for the syncope. The temperature was at first reduced by salicin, but on the 8th day it rose to 39°·5, and kept steadily there; a few spots appeared on the abdomen, and she consented, unwillingly, to be moved from her hotel to a nursing home. The urine showed albumen all through the feverish period, and was partially suppressed to half a litre per day, so that diuretin had to be given. The temperature became normal for the first time

on the 26th day, and the diarrhœa, which had never amounted to more than five motions daily, then ceased. Pleurisy on the right side was discovered on the 26th day, though the patient was too delirious to say whether she suffered pain. On the 30th day, though the temperature was still normal, a thrombus was evidently present in the left femoral vein, and there was so much cystitis, that the bladder had to be washed out. The first relapse began on the 33rd day, and lasted till the 47th, reaching the same height of fever as the primary attack. The temperature was subnormal from the 47th to the 54th day, when it suddenly rose with a rigor to 39°·6 a.m. and 39°·2 p.m., without any apparent cause. The temperature then remained again normal, or subnormal from the 55th day to the 77th, when another definite relapse took place, lasting till the 89th day, after which the patient convalesced with no further difficulty. I gave this patient, as is often my habit, quinine in 30 centigram doses (5 grs.) by the mouth every morning, when the temperature was declining, but in the latter half of the last relapse I gave her intra-muscular injections of 1 gramme of quinine daily. There was no suspicion of malaria in the case, and I do not know whether the quinine did any good. It is popularly supposed that old people cannot get enteric fever, which is, of course, a mistake, but this case is interesting as showing the number of relapses which a delicate woman of 57 can live through.

Three of the patients whom I am reporting had previously had a definite attack of enteric, 20 years, 15 years, and 14 years earlier. Their previous experience did not seem to modify the severity of the second attack.

Mortality.—Seven of my 91 cases died, or 7·7 per cent, but in none of them could an autopsy be obtained. The first case was a lady of 45 years, who died on the 36th day of the disease, being the 15th day of a relapse. This patient was travelling on a Nile dahabia during the illness, and was only seen by me from time to time. She was suffering from great nervous prostration in consequence of the death of her favourite daughter a few days before her own illness began. Another case was a lady, aged 29, who miscarried on the 6th day, and who also died on the 36th day. Her temperature was normal on the

35th day for the first time after a relapse, and everything was doing well with her, until she got a great fright and nervous shock, owing to a family complication. Her temperature suddenly shot up to 41°·2, and she died the same night. The third case was the ambulatory nurse already mentioned, who was extremely difficult to treat throughout her illness. When she was compelled by nurses to keep her bed, she relapsed into a very apathetic state, from which she was unwilling to be roused to take food. In spite of careful nursing she had some superficial bedsores, which came early in the case, and very easily. Her temperature remained between 38° and 40°·7 until the 40th day, when she died of double pneumonia. For the last ten days of her life she had 1/5 albumen in the urine, and diarrhœa, varying between six and nine motions daily; her pulse, during these days oscillated between 126 and 142, and the respirations between 40 and 51. She had a history of former malaria at Mentone, and used to get rigors followed by sweats every afternoon until I gave her one gramme of quinine every morning, besides phenacetin and cold baths.

The fourth case was a very delicate lady, aged 30, who died on the 37th day during a relapse, accompanied by peritonitis. Her temperature, after many cold baths, came down on the 25th day, but the diarrhœa remained, so that she generally had 7 or 8 motions a day. Her pulse rate, during the peritonitis, varied between 170 and 178.

The fifth case was a Coptic boy, aged 5, whose temperature until the 26th day, when he died, varied between 39° and 40°·5. Until the 17th day he had constipation, requiring enemata, but then diarrhœa began, until it reached 17 motions in the day. I began attending him on the 9th day, when the pulse was already 132, it gradually increased in frequency until it reached 192, the respirations rising from 32 to 50. He had well-marked typhoid spots on the abdomen, and lived in an insanitary house, from which his father refused to have him moved; he was very well nursed by two French "sœurs de charité", and kept alive for the last three or four days of his life by caffein and strychnine injections hypodermically. He was essentially one of the cases called by the Germans "nervous fever." Meningeal symptoms came on early, hyperæsthesia, contracture, stiffness of the neck, intense headache and Kernig's sign, with grinding

of the teeth and delirium. He kept his teeth tightly clenched for several days, and had to be fed through the nose. (Kernig's sign is said by Carrière to be present in many severe cases of enteric between the third and 16th day of the disease).

The sixth was a delicate girl of 19, who caught the disease while travelling, and was assured by a foreign doctor on the steamer that her fever was not typhoid, and that she should walk about to get rid of it! When she reached Cairo, I at once put her in a nursing home, where she had severe melæna, and died on the 27th day of peritonitis and a probable perforation. She had a personal predisposition to enteric, and had lost her only brother and several other relations in a similar way.

The seventh case was a lady, aged 30, who did not come under my care until three days before her death. She was free from fever, convalescent and arranging to go to England, but she died suddenly, without any warning, five minutes after the nurse had left her room; there was no evidence of thrombosis in a vein but I think the cause of death was a clot of blood in the heart.

Six of these seven cases were English ladies.

COMPLICATIONS AND SEQUELÆ.

Circulatory system.—Melæna occurred in 5·5 per cent of the cases, being a high average. It appeared on various dates, between the 24th and 44th days, and though always an alarming symptom, it is not necessarily a dangerous one. In one case it occurred on the 13th day, but that patient was obliged to be moved from an insanitary house to another two miles away on the 11th day, and though I have always been careful about moving patients on wheeled stretchers, I think this early hæmorrhage must have been occasioned by the transport. One case had hæmatemesis on the 18th day.

One woman had thrombosis of the right femoral vein on the 28th day, followed by similar symptoms on the left side on the 52nd day, and another woman had a left femoral thrombosis on the 30th day. A girl had severe pain and rapid enlargement of the spleen which I took to be acute splenitis on the 47th day, at the beginning of a relapse.

Digestive system.—Tympanites is a constant symptom of all

cases, but an extreme condition of it, or meteorism, occurred in many cases, producing great anxiety. Peritonitis was present in three cases between the 21st and 31st days, and a probable perforation took place once. Hiccough is a most distressing symptom, which I have twice met with. In one case it lasted incessantly, day and night, from the 12th to the 20th day, in spite of every remedy that any of us could devise. The man was so noisy in his unconscious hiccough, that he drove away all his neighbours from the corridor of his hotel. Vomiting only happened in one case, and I have never found that nausea is a usual symptom; on the contrary, patients gulp down disagreeable mixtures of stimulating food and alcohol with great indifference. The pangs of hunger are only felt by those convalescents who have undergone a mild case, for after a long bout of fever the individual is too exhausted to be hungry.

Respiratory system.—Bronchitis and hypostatic congestion to a mild extent I look upon as symptoms, but four of my patients had double broncho-pneumonia, of which one died. One case, as I have mentioned, developed acute pleurisy on the 26th day, and two others were wintering in Egypt for tubercular lungs and pleuræ, which fortunately remained quiescent during the enteric attack.

Nervous system.—Six patients suffered from rigors on various days between the 19th and 54th. They are always a bad sign, and one of the manifestations of auto-infection in specially septicæmic cases. One very long and dangerous case had no less than 23, having often two or three long rigors in the day. Three children had peripheral neuritis of the lower limbs during convalescence, which lasted two or three months, one child had meningitis, as I have described under the fatal cases. One patient suffered from loss of memory, terror when approached, and fears of persecution, ending in a temporary melancholia.

Genito-Urinary system.—Menstruation generally causes the temperature to fall during fever, like any other hæmorrhage. Three of the patients were pregnant, but only one miscarried.

Two women and one man required the use of the catheter for retention, and two others suffered from partial suppression of

urine during several days. Albuminuria during the febrile period is of no importance, but two cases developed a temporary nephritis after the fourth week.

Cystitis began in two cases on the 20th and 30th days. It is only wonderful that this does not more often occur, if one remembers the predisposing causes, such as, the recumbent supine position for many weeks, the presence of numerous bacteria about the urethral orifice, the bacillus coli communis in particular, favouring an infection of the bladder from the urethra.

Two patients developed superficial bed-sores, for which the nurses were not to blame, and one suffered from an abscess during convalescence.

Among rare complications, I may mention one case of malaria, another of gout, which occurred during convalescence in an American man on the 36th day, and one unfortunate patient who suffered from catarrhal ophthalmia, which began on the 16th day, and polyarthritis which lasted from the 18th to the 47th day. The joints affected were both knees, both ankles, one hip, one wrist, both elbows and one shoulder. None of them required surgical interference.

PATHOLOGY AND MORBID ANATOMY.

Though there is a definite local primary lesion, enteric fever, in spite of its name in England, must not be regarded as only intestinal, but as a form of septicæmia, because we now know that the bacilli pass into the blood, and thence into the various organs, so that the severity of the disease need not necessarily bear any relation to the number and character of the intestinal ulcers. Since the distribution of the bacilli in the body has been known, many enigmatical symptoms have been made clear. The rose spots have been found to contain bacilli, these account for the enlargement of the spleen, and as we are now told that the kidneys are doing their best to eliminate the poison, it is not wonderful that they, and sometimes the bladder break down in the effort. The diarrhœa is apparently caused by a general catarrhal condition of both small and large intestines, which probably provides the best medium for the cultivation of the bacillus. In the constipated cases we must assume that this catarrhal condition does not exist, but I cannot remember ever

to have seen an autopsy on a case which had been constipated throughout the whole illness. These cases are certainly less severe and though, among my private patients, diarrhœa and constipation were equally numerous, seven of the former died, but none of the latter.

By the courtesy of several English military colleagues, I have had the opportunity of attending many post mortems on English soldiers, and have seen some of the intestines coming from Egyptian soldiers who had died from this disease. The post mortem appearances are, as one would expect, exactly similar to those one sees in Europe.

While studying the question as to whether Egyptian infants might possibly suffer from abortive and unrecognized attacks of enteric fever, I found that the intestines of many of them, who had died of diarrhœa but without history of any continued fever, showed unduly raised Peyer's patches, but these were not specially near, or more numerous about the ileo-cæcal valve, and the solitary glands were not apparently enlarged. Moreover, three cases, which were examined immediately after death, showed no typhoid bacilli in the Peyer's patches, but, in order to get an independent testimony, I took some samples of these children's intestines to London, where pathologists, well acquainted with typhoid lesions in children, pronounced, without hesitation, that they showed no evidence of that disease.

DIAGNOSIS.

A continued fever with a morning record of about 39°, especially when accompanied by headache and an absence of other important symptoms, must always be assumed to be, and treated for, enteric fever. I have already laid stress on the fact that constipation is as often present as diarrhœa, and the yellow stools, which are supposed to be so typical of the disease, are, I think, of very little use in diagnosis, for if the patient, during the period of doubt, has been properly fed on a liquid diet of milk, the fæces will be yellow whether the case is one of enteric or not. I have, however, found that a peculiar liquid light brown stool is very suggestive of typhoid. Until complications arise, there is generally no leucocytosis in enteric, and this, in doubtful cases, becomes a useful point.

The absence of spots is of no value in coming to a diagnosis, but their presence, if typical, is practically final. A conclusion may often be arrived at by the exclusion of any other cause of the fever, but a certain history of a previous attack will make one hesitate to diagnose enteric fever.

Of 1159 consecutive admissions to the Edinburgh infectious hospital, all of which were suspected to be enteric, 347, or almost one third of them, proved to be other diseases. The percentage of errors is instructive: acute lobar pneumonia 21, influenza 16, tubercular disease 11, broncho-pneumonia 11, constipation 9, diarrhœa 6, typhus 5, and a dozen other diseases 21.

Pneumonia is only difficult to diagnose in those cases where the physical signs are atypical and late in appearing, and where the patient suffers early from delirium and the "typhoid state," it must not be forgotten that enteric does not start with the abruptness of pneumonia, and that herpes on the lips is as common in the latter as it is rare in enteric.

Influenza, especially if without a sudden onset, and accompanied by headache and diarrhœa, I have often found most difficult to differentiate for at least a week. If an influenza epidemic is prevalent, one can only suppose that the more likely of the two diseases is present, and I have often believed that an influenza case with a steady temperature of 39° to 39°·5 was one of typhoid, until at the end of the first week enteric spots, or influenzic broncho-pneumonia have settled the question, and, as a minor point, I may mention that the influenza patient will often have a more thickly coated tongue, and a positive loathing for food.

Tubercular meningitis, for some unknown reason, is rare in Egypt, yet tubercle is more likely than enteric to be met with in a native, but the reverse is the case amongst Europeans in Egypt; and in tuberculosis, when there is any local inflammatory mischief going on, there is always a certain degree of leucocytosis, though the serum test may prove the only method of diagnosis possible.

Malarial fever can be excluded by the absence of the hæmatozoa in fresh specimens of blood taken on two days; in Egypt enteric is the more probable of the two, but in many parts of the Sudan it is the less likely. It is more common to mistake enteric for malaria than to diagnose a case of malaria as typhoid.

Mediterranean fever is a disease of cities, and not of camps or deserts, it is hardly known in inland Egypt except for the sporadic cases imported from Europe. But in coast towns, such as Alexandria, Port Said and Suez, the two diseases are both found, and differentiation of doubtful cases can only be made certain by reaction against serum with artificial cultivation of the *Micrococcus melitensis*. Some cases of *Malignant Endocarditis*, and other forms of *Pyæmia* with long continued fever caused by deep seated suppuration, without rigors or sweats, extraordinarily simulate enteric, but the presence or absence of leucocytosis is an important aid.

Appendicitis, like enteric, is very rare among natives, and very common among Europeans, but the two diseases may easily be confounded at the beginning from the occasional similarity of the fever and intestinal symptoms; many cases in America of enteric have been operated upon under the belief that they were acute appendicitis.

Typhus is not, in my opinion, very like enteric, and after the first week it should not be confounded, until the comatose stage is reached.

Widal reaction.—This serum test is by far the most useful that we have to diagnose enteric from all other diseases, but a good clinician will soon learn to believe its positive results without putting too much faith on negative reports. In employing it, the time limit and the degree of dilution are of great importance, and should be stated when cases are quoted. Undoubted cases will show immotility and clumping in 15 minutes, but the usual time limit is one hour; any changes taking place at the end of one hour may be disregarded. The dilution varies from 1 in 20 to 1 in 1000, but if the reaction is positive only with the 1 in 20 test, you must certainly repeat the examination a few days later. A safe standard is the one employed in Egyptian laboratories, a dilution of 1 in 40. Unfortunately in some cases the positive reaction is not obtained until the second week or later.

Isolation of typhoid bacilli from the blood.—The hypodermic puncture of a vein in the arm or forearm causes little or no

pain, and this method should be used when the Widal reaction is not positive. The bacillus has thus been found at times varying from the 5th to the 20th day, but not later. The method is simple, a culture is made of 2 to 4 c.c. of suspected blood in about 300 to 500 c.c. of broth, a positive report can generally be given in one or two days, but it is not safe to give a negative report until the 5th day of the culture.

It is not justifiable to obtain blood by puncturing the spleen.

PROGNOSIS

Enteric is always a serious illness, and as a relapse may be even worse than the primary fever, no patient can be considered out of danger until his convalescence is complete. The decade from 15 to 25 years not only provides the greatest number of cases, but also the chief number of deaths. Women die more often than men, and pregnancy and the puerperal state add a special danger. Circumstances which influence the death rate are a family disposition to the disease, the quality and quantity of the poison, the time at which the patient comes under skilled nurses, and the treatment by the physician who can never be too experienced to treat this many-headed complaint. Death is generally due to asthenia or to intercurrent affections or to accidents of the intestinal lesions, such as erosion of a large blood vessel, or perforation of one of the many ulcers. The treatment is seriously handicapped when a patient is living in an insanitary house from which the friends will not permit removal. The earlier the case comes under treatment the better for the patient, and there is hardly any disease in which it is more important to substitute the best professional nursing for amateur relations. Rich private patients, who can afford every luxury, stand a better chance of success than others, and I am sure that many of my patients would not now be alive if I had had to think on their behalf of the question of finance. The outlook is bad in the aged, in drunkards, in soldiers on a fatiguing campaign, or if the fever is complicated by existing heart disease. The early appearance of nervous symptoms is always a bad sign, as also are very quick pulse, continued temperature higher than 40°, convulsions, delirium, incontinence of

urine and fæces, and, of course, melæna and perforation. About one fourth of all deaths from typhoid are due to perforation.

TREATMENT

All doubtful cases of continued fever should be treated as enteric until they are proved to be something else. Strict confinement to bed, a compulsory use of the bed-pan, and liquid diet, all of vital necessity in the treatment of early typhoid, will do no patient any harm, even if he, eventually, proves to have only influenza or malaria. The typhoid patient should be given as much air as possible, and therefore be moved into the best room of the house, with windows always open, the ventilation in summer being supplemented by an electric fan. He must be removed from the house in which he has developed the disease, unless it is quite sanitary, and comprises all the necessary conveniences for a long illness. No one can foretell the length of the campaign, and a careful general will make all his dispositions against the enemy on the assumption that it will not be a short one. If it is judged that the patient will require to be moved at any time during the illness, it had better be done at once, before the ulcers have had time to form. Whenever I have actually diagnosed enteric I have insisted on a wheeled stretcher or ambulance waggon being used to transport the patient, but I have seen many moved during their first week in an ordinary carriage without apparent harm. A lady was once taken from a hotel to a nursing home in Cairo, and by some error she did not travel in the stretcher I had provided for her, but was carried in a sitting position in a basket chair. I accidentally met her in the road, when she had completed half the distance and while I was walking anxiously beside the chair, she suddenly slipped out of it on to the road, when I fortunately caught her in my arms, yet though the case was a long one, no ill seemed to be caused by this misadventure, which occurred on the 14th day.

I am encouraged to preach the necessity of absolute rest for the patient, who should allow the nurse to do everything for him, partly because I have had the opportunity of watching the practice of some foreign colleagues, who not only permit their

typhoid cases to get out of bed for toilet purposes, but even make them sit up in bed at the daily visit, that the stethoscope may be applied to the bases of their lungs. I do not want to be too critical, but I am aware that the mortality rate of these practitioners is greater than that of the English and American physicians who are horrified at such proceedings.

The physician's chief duty must be to constitute himself a visiting superintendent of the nurses engaged, and next to watching with a jealous eye every detail connected with the patient's state, he must take care that the nurses themselves are kept in good bodily health by a sufficiency of fresh air and sleep, and he must be prepared to exchange them, or to double their numbers when he finds they are dispirited by a case which is dangerously ill. The patient will not require milk oftener than every two hours by day unless he is unable to drink six ounces at a time, or becomes exhausted between meals, and nurses will often have to be warned that sleep is better than food for a sick man, if he is neither delirious nor comatose. The quantity of milk to be ordered can only be judged by inspecting the stools daily, by which method we also know when the milk should be peptonized, and how much it should be diluted with lime water, rice water, barley water, or mineral waters. Beef-tea, Valentine's meat juice, Brand's essence etc. are all valuable stimulants but not food, and I agree with the nurses that these are apt to increase diarrhœa when it is present. The patient should be encouraged to drink plenty of water (or barley-water), at least 1 or 2 litres a day, as it is a means of washing out his kidneys, which require more help in this disease than is sometimes recognized. I am not in the habit of giving arrowroot, Benger's food, custard or eggs until a few days after the temperature is normal at night, and if there is any relapse, the patient must go back again to his fever diet. Pepsin, nitro-hydrochloric acid or citrate of soda may be given in water to help the digestion of milky food.

Stimulants.—Brandy or whisky are the best, but if the patient is a very rigid Mussulman, ammonia and ether may be substituted. In mild cases no stimulant is necessary, in others they need not be given during the first week or two, or while the pulse is below 120, but as the pulse rises, they should be gradually increased until a maximum of 7 ounces (200 grammes) is

reached; I have, however, in some exceptional cases, given as much as a bottle of brandy a day, when it was necessary to keep the patient alive during the lysis.

Alcohol is also indicated for delirium, sleeplessness and when the first sound of the heart is feeble.

Drugs.—Not even the best nursing will prevent a relapse, and no treatment will shorten the duration of the disease. Drugs are only of use to counteract symptoms, and I have small faith in their antiseptic action, for even if it were possible to kill the bacilli in the intestines, we are unable to reach those in the mesenteric glands and spleen. Salicin, aided by ice-bags, sometimes helps the initial headache, while antikamnia, or phenacetin will relieve the general pains of the body and procure sleep. Antifebrin and antipyrin should, I think, never be used, because the last thing we want to do is to distress the heart. Quinine has no effect in modifying an attack, but it may be used at the beginning or end of an illness when malaria is a suspected concomitant, after the blood has been examined. When a case, during the lysis, has a drop in the morning temperatures without any corresponding abatement of fever at night, I often give quinine for a few mornings as it seems to beat down the afternoon fever even when malaria is absent. Provided there is no great amount of diarrhœa, I think one dose of 3 to 5 grains of calomel is permissible during the first week, but I would never dare to give it at intervals or later, as is sometimes done by foreign colleagues; constipation only requires enemata, and I think these are better given every day than less often; linseed tea sometimes answers better than soap and water. Urotropin, 60 c.gm. three times a day should be given in plenty of water for at least a week in every case after the lysis.

Dr. Caiger, after 16 years experience in fever hospitals, recommends the essential oil of cinnamon, with which he has treated 147 cases, with a mortality of 9·5 per cent. The favourable effects which he noted attending the administration of the drug were: (1) The temperature in the majority of cases ran at a lower level than usual, (2) the patients remained for the most part drowsy throughout their illness, so that mental rest was secured and delirium was less frequent, (3) abdominal pain, dis-

tension, and fœtor of the motions was controlled to a striking extent, and among the 147 cases treated in this way there was no single instance of meteorism. The dose recommended is from two and a half to five minims of the essential oil, given every two hours from the time the case first comes under treatment until the temperature has become normal. The system soon becomes saturated with the cinnamon, as evidenced by the breath, skin and fæces, but not by the urine.

The mouth and nose should be kept clean with Listerine or boracic lotion. Bed sores must be prevented by strict cleanliness and by washing the back and hips at least twice a-day with soap and water, followed by rubbing alcohol on to the skin with the hand, after which the skin should be dusted with a powder made of equal parts of oxide of zinc, starch and boracic acid; on the first threatening of a sore a water bed must be employed.

Meteorism can be temporarily benefited by passing very gently a long rectal tube to let off some of the gas in the lower intestine. Melæna requires such rigid repose that patients should be encouraged to pass all their motions into the draw sheet, on to masses of cotton wool.

Hypodermic injections of strychnia should not be neglected when alcohol by the mouth is not sufficient for the failing heart.

There are several ways of trying to disinfect the intestinal canal, one of the best is to give every four hours a mixture containing the three sulphocarbolates, zinc 0·12, soda 0·12 and calcium 0·18.

The lives of several patients have been saved after perforation by prompt laparotomy and suturing the damaged intestine, with the aid of morphia and cocaine subcutaneously, but to ensure success this bold operation must be done in patients who are not moribund, within twelve hours of the perforation. In 1901 Dr. Creswell successfully operated on a British seaman at Suez, in spite of a creeping bubo in the right groin; the ulcer had perforated about the 21st day, and though the man had two relapses afterwards, he eventually recovered. In the same year he again operated on an English telegraphist, whose ulcer had perforated at the end of the third week; the patient survived ten days, but then succumbed to a second perforation

just above the sigmoid flexure. At the autopsy numerous ulcers were found in both the small and large intestines. The medical treatment of perforation has hardly ever saved a patient, but laparotomy has, since 1889, already saved 95 lives. There is, therefore, when a competent surgeon is at hand, no contra-indication to this operation save a moribund condition of the patient. The most common site is in the last 12 inches of the ileum, but if no perforation is found in the ileum, cæcum or appendix, the next most likely place will be the sigmoid flexure. It must not be forgotten that the perforation is usually single but may be double.

The operation consists of finding and closing the perforation without stopping to pare the edges, of emptying and cleansing the peritoneal cavity, and of establishing and maintaining proper drainage.

Saline infusion of twenty to thirty ounces into the areolar tissue of the chest or thighs is of great value when a patient is sinking from heart failure in spite of strychnia given hypodermically and alcohol. It is of no use to pour indiscreet quantities of food or alcohol into a stomach which is not responding. At autopsies of well nursed enteric (and other) cases you will find the stomach inconveniently full of liquid food, this probably impedes the action of the already labouring heart.

Cold baths.—Osler says that about 75 per cent of typhoid cases recover under any form of treatment, and even without the good nursing and regulated diet upon which we lay so much stress, but by judicious care, by careful feeding, and by the withholding of drugs of uncertain value, 15 additional patients in each hundred are saved, and, apparently, an extra 3 or 4 per cent are saved by hydrotherapy. The cold bath can only be carried out in a well-appointed hospital or in a private house where there is no lack of nurses. Anyone who has seen a muttering, delirious patient suddenly raise his head, smile and talk with the attendants one minute after he has been immersed in a full-length bath, cannot doubt the temporary advantage of reducing the fever.

This method of treatment has also a good effect on the heart, lungs, kidneys and skin, especially if the patient be rubbed all

the time he is in the water. But unfortunately the bath must be repeated so often, that both patient and nurses are apt to get worn out. In order to try and diminish the number of the baths, I give them only when the temperature has reached 40°, and do not repeat them for at least three hours, seldom giving more than four in the 24 hours. Another useful modification is to put the patient into a bath at 25° C., and reduce it rapidly by ice to 20°. In cases where the bath is contra-indicated, or unattainable, the cold wet pack may be used under medical supervision, or the body may be uncovered in sections and sponged over with ice cold water. The blueness and shivering which often occur after these methods of treatment are of no importance. The water bed is another very useful way of lowering the temperature, and has the advantage of not moving the patient; cans of iced water being substituted for the tepid water in the bed.

After the second week the physician should be on the watch for possible perforation, and should examine the abdomen at every visit.

The nurse too should be warned to give early information of pain, vomiting, hiccough or collapse, or if there is any sudden change in the rate or character of the pulse and respiration.

Anti-typhoid-Serum.—Chantemesse of Paris claims that the typhoid death-rate can be greatly reduced by employing serum in addition to cold baths and the ordinary symptomatic treatment. His figures are certainly worth quoting. During 20 months in 1901—2 there were 1478 typhoid cases treated in 15 different Paris hospitals, which gave the high mortality of 19·3 per cent, while during the same period 186 cases were treated by him in the Paris hospital *Bastion 29*, with only 3·7 per cent of deaths. One would like to know whether the proportion of nurses to sick is the same in all these hospitals, but it is claimed for the serum that it alone has caused this great reduction in the death rate. The serum does not, however, prevent perforation, for death occurred in five of the cases from this complication.

As to the precise nature of this serum, there is at present some uncertainty, though its success in practice is, apparently, very striking. At the seventh French Medical Congress held in October, 1904, in Paris, it was stated that now 696 cases have been treated in this way by Chantemesse, Planté and

Foucauld, with a fatality of only 5 per cent. It appears that a horse is immunized by repeated inoculation with toxic products of the typhoid bacillus obtained by special methods of cultivation and that the serum eventually acquires curative properties, but the process requires many months for its completion.

PREVENTION.

Medical men and the public must be taught that precautions are just as necessary in the case of enteric as in that of cholera, yet in Egypt the latter inspires great, and often unnecessary alarm, while the former is treated with profound indifference. Every town and village should be supplied with a pure supply of drinking water, and the Sanitary Department, which since 1884 has already done so much in this direction, will, it is hoped, continue to have this ideal before it. Previously to 1891 Europeans in Egypt trusted for the filtration of their domestic supply to native filters (ziehs), which remove mud, but cannot stand any bacteriological test. But now, for some years, the best houses, barracks, hospitals, schools and prisons have been supplied with Pasteur-Chamberland, or Berkefeld filters, and it is to be hoped that the use of these will gradually spread into native dwellings, and help to lessen the great mortality among children from diarrhœa. Doctors, who are supposed to be educated guides through the mazes of ignorance, are very negligent about seeing what manner of water their patients drink, and whether the European filters are ever kept clean.

As regards ice, the public must put its faith in the Sanitary Department, which is morally responsible for the inspection of ice and mineral water factories. The same applies to milk and all dairies, the law at present allows inspection of all native cowsheds, and these are, therefore, kept fairly clean by the visits of veterinary inspectors, but there is no similar law for European dairies, and no organized inspection, though many of the owners profess that they are willing to submit to voluntary inspection, and even to pay for it. It is obvious that, as regards this one disease, European dairymaids are more dangerous to the community than Egyptian milkers. Careful householders must continue to sterilise or boil the daily milk directly it reaches

them, and the system followed by some English people of sending a child to a tea party with its own bottle of milk is to be commended. My own children in 1899 caught enteric at a friend's house in Cairo by neglecting this precaution. The oyster beds of Alexandria and Port Said should certainly be under sanitary control, and all salad and vegetables, which are to be consumed raw, should be washed several times in the consumer's kitchen in filtered water.

It is not enough to supply towns with a pure water supply, for experience shows that enteric cannot be diminished until some system of drainage is adopted. Dantzig was supplied with new water-works in 1869, but a high death rate from typhoid persisted until the introduction of a sewage system, after which it fell from an average of 9·9 per 10,000, to 1·5. Again, in Stockholm the enteric mortality fell step by step with the increase in the number of sewers, falling in ten years from a mortality of 5·1 per 10,000 and 8937 metres of sewers to 1·7 deaths with 65,709 metres of sewers, and similar experience might be quoted from many other towns. As an instance of the length of time that typhoid stools may remain dangerous in a cemented cesspool, I may mention a case which occurred at Schilligheim. Typhoid stools, without being disinfected, were for five days emptied into a latrine, five months later the cesspool was emptied, and the contents spread over the garden, which is a common custom in Germany; after exposure to the fresh air and to the cold of February during fifteen days, samples of the garden earth were found to contain virulent typhoid bacilli, which were, of course, dangerous for anyone eating raw vegetables from that garden.

As it is impossible for the Egyptian Government, hampered by finance, to do everything, and as there is a very proper feeling among the English rulers that available resources should be expended chiefly upon ameliorating the condition of the natives, rather than the Europeans, I have tried at various times to induce householders in Cairo to try and protect their families and their dwellings by creating a Sanitary Association similar to those which have done good work in England, and I still hope that this idea may yet be carried out.

Food must be protected from dust and flies, and all clothing

and bedding should be disinfected by steam at one of the stations where the Sanitary Department does this work gratuitously.

The ideal method of disposal of fæces is to burn them, but this is not often practical. The dry earth or pail system is a danger in tropical climates, unless all the excreta could be disinfected. There are many ways of disinfecting typhoid stools, but the simplest and the cheapest is obviously the best. For many years I have recommended the method first taught me by Professor Koch, of mixing quicklime in 5 parts of water to make milk of lime, and then adding to the excreta a quantity of this milk of lime equal to their bulk. This is found to destroy the bacilli in less than half an hour, after which they can safely be thrown down the drain pipe into the cesspool. On military service, when boiling and burning are both impracticable, deep burial of fæces must be recommended, after treatment if possible with disinfectants.

Enteric patients should be treated in special wards in a hospital, and not mixed up with other cases, which is so often done. The urine and sputa, as well as the fæces, must be disinfected as soon as possible. Those engaged in nursing require special protection, and even the most experienced should be reminded to scrub their hands and nails in a disinfectant after touching the bed-pan, linen, etc. Thirty years ago I remember a fellow-student who always retreated from the neighbourhood of a typhoid bed when the sheets were being disturbed, lest he should be infected by contaminated dust from the patient; we all ridiculed him then, but I should not do so now.

It is unfortunate that doctors in Egypt do not yet give the Government all the help they can by notifying infectious diseases; they are liable to be fined if they neglect to do so, but perhaps the system will work better when the Government finds itself in a position to pay them fees for notification, as in Europe.

Preventive Inoculation.—Since 1896 anti-typhoid "vaccine" has been experimented with in England, India, South Africa and Egypt. Many of us are not yet wholly converted to its value, but its supporters maintain that statistics are greatly in its favour. In 1903 the College of Physicians in London appointed a Committee to consider and report on the safety and prophylactic value of

Dr. Wright's inoculation. I cannot do better than quote their conclusions: "After careful scrutiny of the statistics from both official and private sources, which have been made available, we are of opinion that not only is a lessened susceptibility to the disease brought about as a result of the inoculations, but that the case-mortality is largely reduced. We are further of opinion that with due care the process of inoculation is devoid of direct danger, but that under special circumstances there may possibly be some temporary increase of susceptibility to infection immediately following inoculation; and it is, therefore, desirable that the preparation of the vaccine and the inoculations should be carried out under specially skilled supervision." One of the questions which requires settling, is whether the inoculation is to be single or to be repeated a second time in a fortnight. The inoculation affects people in different ways, and it is most unwise to allow the inoculated to walk about for at least twenty hours after the operation. At the time of my own inoculation I saw syncope attack strong men who had neglected this advice. The usual symptoms are local inflammation over a patch of about four inches diameter, faintness, restlessness and fever varying from 38°·5 to 40° C.

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MEDITERRANEAN FEVER

Synonyms.—Malta fever, Gibraltar fever, Rock fever; Neapolitan, Cretan, Cyprus, Danube, Levant fever; Undulant fever.

The above terms are all still used, while the following have been employed of late years though they have nothing to commend them: Gastric remittent, bilious remittent, intermittent typhoid, typho-malarial, fæco-malarial, sewage, cesspool fever.

Definition.—An endemic, specific fever, sometimes becoming epidemic, of long, indefinite duration, and a tendency to long, wavy relapses; characterized by constipation, profuse sweats, anæmia, debility, neuralgia, and sometimes rheumatic sciatica or swelling of joints. The disease is dependent on a specific microbe, but is not directly contagious, and is attended by a low mortality.

History.—Hippocrates wrote of long fevers occurring on the coast of Greece with as many as four lengthy relapses, and on one occasion death on the 120th day, but it was not until 1859 that this disease was first described as a specific illness. In 1886 Bruce found the *micrococcus melitensis*, which he named after the island of Malta, but, unfortunately, the disease is still much confounded with malarial and enteric fevers.

Distribution.—Besides the places which have given this fever the above mentioned names, it is known in other sea ports of Italy, in Sicily, Sardinia, Constantinople, Tunis and Algiers.

But recent experience has shown that "Mediterranean fever" is by no means a sufficient name, for it occurs on the Red Sea coast, at Suakin, Massowah and Zanzibar; in India, in the Punjab, at Simla, Delhi, Agra, Allahabad, and in Assam; at Hong Kong, Porto Rico and in the West Indies and Philippine islands.

This fever was never recorded in Egypt until I recognized a case in November 1883 in a young English lady who had travelled leisurely through Italy on her way to Cairo. Her symptoms were similar to the disease which I had had opportunities of studying in Gibraltar and Malta, quinine in doses of 40 grains daily had no effect upon the temperature, which lasted for 100 days, and an additional argument against its having been enteric is that the patient underwent an attack of the latter in Cairo a year later. Since then many English soldiers have brought the disease with them from Malta and Gibraltar and have been treated in the military hospitals.

In 1897, for instance, two battalions from Malta furnished eight cases in Cairo and five in Alexandria. During the last four years two residents of Cairo have developed the disease in Egypt on their return from England in the autumn.

But the Egyptian record is not confined to imported cases, for occasional sporadic instances occurring in the country have been seen among Europeans at Port Said, Alexandria and Suez, and Mr. H. Milton saw a case (proved by the serum test) in an Egyptian who had come from Mit Ghamr to Cairo, and had never in his life been out of Egypt or even as far as Alexandria.

Since then, in the autumn of 1903, the blood of a native child in Cairo showed a positive serum reaction to 1 in 200, and by puncturing the spleen Dr. Dreyer found the *micrococcus melitensis*, and made a culture from it, while, in the spring of the same year, a localized epidemic occurred in the Port Said Government hospital among the resident attendants and servants, all being natives. The average duration of fever was two months and a half, and in two of the 12 cases the micrococcus was found in the spleen by Dr. Bitter.

Captain Stallard, R.A.M.C. saw 12 typical cases at El Obeid, after the rains in August and September 1902, and an English patient has been treated for Mediterranean fever in Cairo, who contracted it either on the White Nile or at Khartum or Berber; his blood gave a positive result with the serum test. Captain Rivers R.A.M.C., while on duty at Kassala, saw during one year four cases among Abyssinian regular soldiers who had apparently caught the fever in the Kassala district; all four cases occurred between June and September,

that is, during or after the annual rains. There is much malaria at Kassala at this time of the year, but hæmatozoa could never be found in these four cases, and they proved quite refractory to quinine by mouth and needle.

An interesting case has lately been reported of this fever in a man who had never left England. He was a gunner, who died at Dover on the 122nd day of the disease, and the serum test proved to be positive on the 68th day.

The statistics of the British Navy for the three years 1901-3 give 831 cases in the Mediterranean, of which 620 were attributed directly to Malta, 32 to Gibraltar, 30 to Corfu, 3 to Alexandria and the remainder to other ports, mostly Lemnos, Platea, Nauplia and Arancia Bay.

Causes.—The disease is due to the entrance into the body of the specific micrococcus, but it is still a moot point whether the infection is air borne or water borne. Possibly, as in the case of enteric fever, both methods are employed. Three predisposing causes are believed to be essential, a sea coast or large river, exposure to sewage and hot weather. It will be noticed that nearly all the parts of the world from which the disease has been reported are on sea coasts or in islands or on the banks of large rivers. It is also well known on ships in various harbours where sailors are exposed to sewage all round them.

There can be little doubt that it is a filth disease, and many believe that the microbe is able to exist in the soil, but is apparently not present in clean earth. The disease has not been eradicated from Malta and other places, in spite of improved sanitation, but this is perhaps due to the fact that the old rock sewers have not been abolished, and the hygienic measures have not been sufficiently thorough.

On the other hand the Rock fever, for which Gibraltar used to be so notorious, is said to have decreased during the last 20 years in the proportion of about 90 per cent. This improvement is apparently due to better sanitation, such as the lengthening of the main sewer, the greater attention paid to house drainage, street paving and scavenging, stricter supervision of food and milk supplies, and, most important of all, the formation of reservoirs in the rock for supplying all the inhabitants with pure filtered rain water.

The season of greatest prevalence is from June to September while the coldest months from December to February furnish the fewest cases. At Malta the cases occur mostly when the rock sewers are undergoing a process of drying after the rain.

Though this fever is apparently not contagious from one patient to another, inoculation experiments have proved that men and animals can be infected through the skin. It has therefore been suggested that it is an insect-borne disease, like malaria.

It occurs most commonly between the ages of six and thirty, and equally affects both sexes and all stations in life, but attacks strangers more than natives. Residence in an infected area does not confer immunity, though this may be gained, at least for several years, by an attack of the fever.

A few facts are known about the micrococcus: cultures kept at 22° C. still retain vitality after 14 months, it lives in milk for three weeks, in tap water for three days, and dies immediately in sea water. In urine it can live from three to six days, and Horrocks has found that it can live in dust for at least 69 days, and on dried clothing for even longer, but exposure to the sun kills it in a few minutes. Experiments of feeding monkeys on the microbe are invariably negative; one man is said to have been artificially inoculated by the conjunctiva. It has been isolated from the urine and the blood, but never from the sputum, expired air, sweat, or fæces. It has been suggested that the disease is spread (1) by the urine, (2) by exhalations from the skin or (3) by infected dust.

Varieties.—There are two types of the disease with which students must be familiar.

(1) The intermittent, which may produce a temperature chart exactly similar to that of a patient suffering from tubercle or deep seated pus, or may show occasional very short waves of pyrexial intensity; and (2) the undulant, which caused Hughes to suggest this name for the disease.

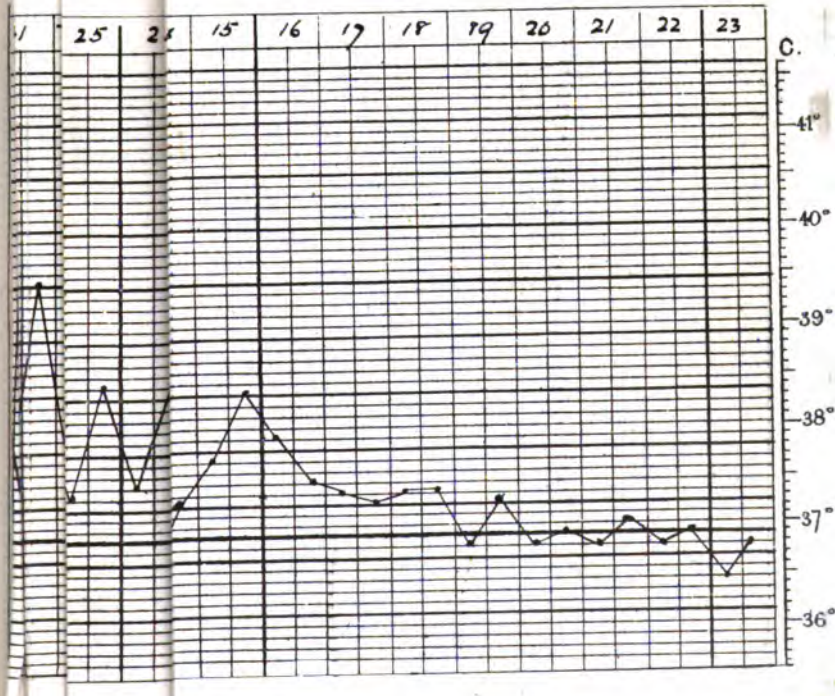
Symptoms and Course.—Obstinate constipation, extreme anæmia, debility and enlargement of the spleen are the only certain symptoms which accompany the fever. The indefinite duration of the temperature distinguishes this from all other fevers, for in severe cases the pyrexia may last for six months and con-

valescence not be completed for two years. The average stay of soldiers in hospital is from 70 to 90 days. The fever is characterised by intermittent waves of more or less remittent type, each wave lasting from one to three weeks with generally an interval without fever of two or three days. The waves may vary in number from one to seven, the average being three. The length of the wave is usually from 10 to 15 days, but it may be as short as three days, or as long as fifty.

The temperature in my cases has generally reached 40°, but seldom goes above it. The highest record on my charts is 40°·5. Many patients develop a tendency to enteritis, but Peyer's patches and the mesenteric glands are never involved. Profuse sweating is often present, and accounts for various names given to the disease by the Italians, such as *febris sudoralis*. Emaciation and loss of hair are necessary results of the prolonged fever.

Complications and Sequelæ.—The most common complications are rheumatoid affections of the joints, such as the hips, knees or shoulders, sciatica and respiratory diseases, including bronchitis, pleurisy and pneumonia. These latter, together with hectic fever and sweats, present a picture which is often suspiciously like tuberculosis. The following cases illustrate the length of the disease, the tendency to relapses, and the fact that the personal equation will modify the poison and produce in two members of a family very different complications.

Case 1.—An English lady, æt 40, stayed at an hotel in Malta from April 6th to 15th, 1896, and then slept for one night in Syracuse before her return to London. About May 1st she felt ill for the first time, but it was not recognised that she had fever until May 19th when she was put to bed, and kept there till July 27th, suffering from insomnia, constipation, want of appetite, profuse sweats, loss of hair, anæmia and emaciation. On May 26th three days of apyrexia began, then followed a week of 38° a.m. to 39° p.m., then six afternoons when the thermometer reached 40°, then a gradual descent of both morning and evening temperatures until June 18th when pneumonia of the right lung occurred, ending by lysis ten days later. The temperature only remained normal for four days and then showed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another wave of fever from July 2nd to July 20th and again a miniature wave from August 1st to 7th. The sixth and last wave on the chart was a serious one lasting from Sep. 15th to the middle of October. The patient remained weak and anæmic in spite of various changes of climate until Dec. 20th, when she had a dangerous attack of acute bronchitis lasting three weeks. She had, until her unfortunate trip to Malta, always been a very strong healthy woman, and had never had any bronchitis before, so that this attack seemed to be a distinct sequela of the fever and pneumonia. In February 1897 she was sent to Cairo, where I found that she was still suffering from anæmia, inability to walk, loss of weight, some lividity of the face and hands, much cough and expectoration, many bronchitic signs in both lungs and dulness at the right base.

In May she was able to return to England quite well, one year after the beginning of the illness, and she has remained in good health ever since. It is to be noted that she never had any rheumatic symptoms. During the fever she had some transient erythematous patches on the skin of the back, sides, abdomen and face.

Case 2.—A boy aged 12, travelled with his mother (Case 1) and was therefore exposed to the poison in Malta or Syracuse, in April. He remained apparently quite well and was not known to suffer from fever until July 18th, when, at school, his temperature was found to be 40°, and there was some vomiting, diarrhœa and urticaria, at first supposed to be due to eating over-ripe fruit. But when the fever continued, and was found to be accompanied by no symptoms, except constipation and an enlarged spleen, the disease, like his mother's, was called Mediterranean fever. His temperature may have been somewhat modified by quinine, arsenic, salicin and phenacetin, but for two months and a half the evening rise ranged from 38° to 39°·5, occasionally reaching 40°, while the morning temperature varied from normal to 39°. The chart showed a continuous intermittent range, with a slight tendency to waves, but without periods of normal evening temperature between them. This continued fever produced emaciation, great weakness and depression of spirits. In September there was an improvement in temperature and gain of strength

but in the middle of October, soon after the temperature had become normal, acute rheumatism in the right knee occurred for the first time as a further confirmation of the diagnosis. Between October and January he had five similar attacks of fever and rheumatism in the right thigh. At the end of February, while on the Riviera, he had an attack of fever for eight days, rising as high as 40°·2, but no rheumatism, and in March he was brought to Cairo to be under my care. His splenic dulness then measured 4½ inches, but his anæmia and constipation yielded easily to treatment, and he was soon strong enough to play golf for half an hour a-day. On April 11th, just a year after his visit to Malta, I thought him well enough to join a donkey picnic in the desert, but this exertion was followed by five days fever, without rheumatism. The fever was the shortest bout he had ever had, and disappeared the day after he took some quinine; I gave the quinine in three doses each of half a gramme at 8, 10 and 12 in the morning, a method which answers well in other fevers when the morning temperature is low. When this patient first reached Cairo I found that his urine was extremely acid, high coloured, and of specific gravity 1024—1026, but without albumen, and I therefore made him drink large quantities of Contrexèville water. During his attack of fever, there was no albuminuria, but for a month afterwards there was always albumen after meals, though none before food. At the end of May he returned to England with a splenic dulness of three inches, without fever, rheumatism or albuminuria, and able to walk several miles without fatigue, and since then he has always remained well. The only reason I can suggest for the great difference in his complication to his mother's, is that he inherited a distinct uric acid diathesis, which his mother did not. I should add that his blood never showed any evidence of malaria.

Morbid Anatomy.—The autopsies in Egypt at which I have been present displayed changes similar to those I have studied at Malta. The spleen is enlarged, hyperæmic and soft, weighing from 11 to 30 ounces. The liver is generally a little large and congested. The lungs usually show hypostatic congestion, and the kidneys are also hyperæmic. The small and large

intestines are congested, often in patches; there is no swelling nor ulceration of solitary glands or Peyer's patches, nor is there in ordinary cases any enlargement of the mesenteric glands.

Diagnosis.—Mild cases, with little fever, are often overlooked and treated at first as dyspepsia or debility. In countries where malaria is prevalent that disease can be negatived by the absence of parasites in the blood, and the unchanged condition after quinine. To differentiate cases from the hectic fever of tuberculosis or liver abscess, we must rely upon local symptoms, bearing in mind that phthisis is not an uncommon sequela of Mediterranean fever. The effusion into joints and neuritis must be distinguished from acute rheumatism, synovitis and neuralgia.

But the chief difficulty in diagnosis occurs in the beginning of a case when enteric is suspected; the milder and longer course of the disease, the absence of eruption, the profuse sweats, the extremely remittent or intermittent type of fever and very often the rheumatoid symptoms help one to diagnose Mediterranean fever, especially if the illness began on some sea coast where it is known to exist.

Of late years serum diagnosis has been of the greatest help, and seldom fails to distinguish this fever from enteric. I am indebted to Major D.V.O'Connell R.A.M.C. for the details of the methods satisfactorily used at Malta for the last five years. Both methods of obtaining serum are equally efficient, but the former is the more rapid, provided a centrifugal machine is available.

(1) A drop of blood is drawn, preferably from the lobe of the ear, into an ordinary vaccine lymph tube, both ends of which are then sealed by heat. On being brought to the laboratory, the ends are broken off, and the capillary tube is put into a test tube about 2½ inches long. Ten or more drops of distilled water are added to dilute it, and the tube is then put into a centrifugal machine which is whirled for about two minutes. The watery fluid resulting from this is the diluted serum required for the test, a drop of which is taken up with the capillary tube and placed on a clean slide. Then,

with a sterilized platinum needle, a very small quantity of the active growth of the M. Melitensis on agar is taken up, and mixed thoroughly with the diluted serum on the slide, the mixing being done with the platinum needle which is subsequently sterilized in the gas flame. A cover slip is then put on the slide, which must be examined with a low power (one sixth).

In some cases clumping is immediately seen, but in others you must wait about half an hour. If the clumping does not then appear no reaction will occur at all, owing either to the case not being one of Mediterranean fever or because a too diluted serum has been used. The least amount of dilution which is satisfactory is 1 in 20, but in most cases a dilution of from 1 in 40 to 1 in 200 will produce the clumping.

(2) A small blister may be made with blistering fluid on the upper arm, about the size of a 10 piaster piece. In twelve hours a large quantity of serum can be obtained in capillary tubes from this, and a drop of it can be diluted and centrifugalized as above, this serum is quite as effective as that obtained directly from the blood.

To obtain the reaction with certainty, the patient should have had the fever for at least a week or ten days. Cases with high temperatures usually clump quickly.

Prognosis.—The mortality does not usually exceed 2 per cent, the majority of deaths occurring during the first six weeks of the attack. The pyrexia averages about 60 days, but may vary from 14 to 300 days. Excessive diarrhœa, vomiting, and concurrent exhausting diseases are all dangerous.

A sub-normal temperature lasting for a few days, accompanied by a perfectly clean tongue, and returning appetite, are the surest signs of approaching convalescence.

Treatment.—The patient must be removed from any insanitary surroundings, and be treated in every way as a case of enteric, until the diagnosis of the latter is excluded. He must be confined to bed so long as acute symptoms persist, and the drug treatment must be entirely symptomatic. The diet should be graduated according to the height of the fever and the condition of the tongue, but it need not be so restricted as in enteric

fever. The rheumatic symptoms will require flannel clothing and cotton-wool, and the patient should sleep between blankets. Open air treatment will help convalescence, and patients should be sent to a warm climate for the winter months, though they should not be allowed to travel during the acute stages.

Prevention.—Sanitary houses and carefully chosen camps for troops are all-important. Special care should be taken to avoid the bites of insects wherever this fever is prevalent. The urine and fæces must be rigorously disinfected, and every care taken with regard to water supply and general hygiene. Attendants on the sick should carefully disinfect their hands.

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SIMPLE CONTINUED FEVER.

Synonyms.—Febricula, Ephemeral fever. This is a term not often employed in civil life, but much made use of by army medical officers. During the last ten years the average number of cases so diagnosed per year at Kasr el Ainy civil hospital is only 11, while in the Egyptian army in Egypt there were 127 cases, and in the Egyptian army in the Sudan the yearly average is as high as 1411. Among the British troops in Cairo and Alexandria 343 cases were returned on an annual average under the heading of "other continued fevers," and I think it is fair to assume that about 300 of these were called simple continued fever. The army medical officer might explain this discrepancy by pointing out that soldiers must enter the hospital if they have any illness lasting more than 24 hours, while a civilian usually stays at home for a short indisposition; also the custom of the English army is to require a written diagnosis soon after a patient's admission, and so all cases without any very obvious cause for fever have a tendency to be labelled under this heading; again, the army doctor is obliged faithfully to follow the official Nomenclature of Diseases, which does not include many of the causes of the soldier's attacks of fever.

Common causes are: exposure to sun or heat, chills, fatigue, irregularities in diet, and those rather mysterious cases to which I have alluded under the heading of enteric fever, where people living in insanitary houses suffer from periodical attacks of pyrexia, which cannot, however, be dignified by the name of abortive enteric.

The temperature may be anything from 38° to 41°, and the most common symptoms are headache, malaise, muscular pains, a white furred tongue, anorexia, thirst, and irregularity of the bowels. The fever usually lasts from three to ten days, and if further prolonged the diagnosis must be reconsidered.

Soon after the British army occupied Egypt, a Principal

Medical Officer, disgusted at the number of times death and typhoid ulcers followed the diagnosis of "simple continued fever," decreed that every case under this heading must have the diagnosis changed to "enteric" if the fever lasted more than three weeks. The diagnosis should, in my opinion, only be permitted in non-fatal cases of a few days duration, when no obvious cause can be discovered. The disease is not contagious, and as patients never die of it, its pathology remains unknown. Many cases occur under this heading which I think might be called Influenza or Dengue. The treatment depends entirely on the symptoms, and should consist chiefly of rest in bed with a very simple diet.

INFECTIOUS JAUNDICE.

Synonyms.—Febrile jaundice, Epidemic jaundice, Septic jaundice, Weil's disease.

Fr. Typhus bilieux, Ictère infectieux fébrile.

Ger. Biliöse Typhoid, Icterus typhosus, Typhus biliosus nostras.

This disease, like many others little understood, has been called by a variety of names, of which, perhaps, Infectious Jaundice is the best. The disease has nothing to do with typhus or typhoid fever, and therefore it is a great pity to continue misleading titles which date from fifty years ago, when many unknown fevers were thought to belong to a typhus group.

Definition.—An acute, infectious disease, sometimes becoming epidemic during the summer months, characterized by fever, jaundice, enlarged liver and spleen, nephritis and some nervous symptoms.

History.—Hirsch records 34 epidemics of jaundice, mostly in Europe, between the years 1745 and 1885.

In 1886, Professor Weil of Heidelberg published four cases of infectious jaundice under the title of "A peculiar form of acute infectious disease characterized by jaundice, swelling of spleen and nephritis." He and other Germans were in doubt whether his cases belonged to a special disease, or to Griesinger's "bilious typhoid," and it was decided to call them "Weil's disease."

Many English and American writers have provisionally adopted this term because it is preferable to "bilious typhoid," and because every one now agrees that the malady has no sort of connexion with relapsing fever nor with the variety of it described by Griesinger.

Most physicians who have seen Weil's disease in Europe and

infectious jaundice in the Mediterranean agree that they are practically identical.

Distribution.—Legrand has reported two fatal cases from Suez, and I saw, in October 1886, an English maid who died of the disease in Cairo, having apparently contracted it in Alexandria. The autopsy was similar to those which I had seen during an epidemic in Alexandria. Again, in January 1899, three cases of dying men were admitted at Kasr el Ainy, and eight others found dead in the street were transferred by the police to Dr. Nolan, for a medico-legal report. The diagnosis wavered for a time between hæmorrhagic septicæmia of unknown cause, and doubtful relapsing fever, though no spirilla could be found in the blood, until I was able to show that the localized epidemic was exactly similar to the infectious jaundice of Alexandria. Since then, Stassinopoulos has stated that he has seen three cases in Cairo between 1895 and 1903 similar to those well-known to him in Alexandria; but, with the above exceptions, the disease seems to be as unknown in Cairo as it is common in Alexandria, where it has been endemic certainly since 1870 and perhaps even longer.

Doubtful cases have been reported from Tanta and Kafr Zayat and, outside Egypt, from Malta, the Ionian Islands, Dalmatia, Athens, Constantinople, and Syria.

In Smyrna the disease has apparently been endemic since 1837, and 185 cases were reported from Nauplia (Greece) during four months in 1886.

In the summer of 1877, during the Russo-Turkish war, several of us, then in the Balkans, were puzzled by an infectious fever with jaundice, which attacked many English doctors and correspondents. In spite of large livers and spleens, the disease was certainly not malarial, and we were obliged to accept the diagnosis of local doctors, who were well acquainted with the malady and called it "Bulgarian fever." I have since concluded that this unknown fever was Weil's disease, for though we were camping near lovely rose-gardens, we were surrounded by the unburied bodies of hundreds of men and animals, and by thousands of wounded Turks in every degree of pyæmia and hospital gangrene. However, we had no possibility of examining the

blood of our patients, so it is possible that they were really suffering from relapsing fever without relapses, though the temperature never ended with an abrupt crisis.

In England, small epidemics have been reported in 1895 and 1901, and a few cases have occurred in the United States, while localized outbreaks have been known in Pekin and Tientsin in 1864-5, and again in 1898-9.

It is quite possible that some of the mysterious fevers masked under the heading of malaria in India and Mauritius, have been really the disease in question, which, I think, merits more attention than it usually receives from English and American authors.

CAUSES

Age.—The most common age in all countries seems to be from 20 to 30, but it may claim victims from 15 to 40. It has never been recorded in Egypt under the age of 15 years, though there have been rare cases in Germany and Greece between 8 and 14 years of age.

Sex.—Many observers have never seen a case in a woman, and I have only seen one, but Valassopoulo's statistics give 144 men to nine women. The only reasons one can suggest for the rarity of this disease among women and children are that they are less exposed to unclean occupations and sewage contamination on a large scale, and also that they run less risk of being poisoned by contaminated meat.

Nationality.—In Alexandria the Greeks seem more predisposed to it than other Europeans.

Occupation.—In Cairo it is as a rule only the lowest classes who are attacked, but in Alexandria during an epidemic it has been seen among bankers, lawyers, merchants and doctors, as well as among bakers, grocers and butchers. In Germany 38 out of 53 cases were in working men engaged in insanitary occupations. At Nauplia the wine sellers headed the list, and I may mention that alcoholic excess seems to be a predisposing cause. Out of 144 men reported by Valassopoulo, there were 18 bakers, 9 grocers, 31 clerks, 17 servants besides 5 cooks, only two wine sellers and no butchers at all.

Season.—The disease occurs in Alexandria between April and

October, but rare sporadic cases may be seen during the winter months. Kartulis believes that there are two periods of maximum intensity, between May and June, and again from mid September to mid October.

Contamination by sewage or putrid meat.—There can be no doubt that this is a filth disease. In Alexandria it has become more common since the introduction of a bad system of imperfect drainage, the bulk of the patients come from the lowest parts of the town, where the drainage is worst and where the sewers empty into the sea. Cases have been traced to frequenters of coffee houses in these quarters, yet the suburbs of the town which are not drained at all are apparently unaffected.

The two Suez cases resulted after exposure to an open drain undergoing repair. Again, at Smyrna the disease only prevails in the most dependent parts of the town where the sewers join the sea, and at Nauplia it has disappeared since iron drain pipes have superseded badly jointed stone conduits. In Germany men have caught it after bathing in a river contaminated by sewage, and after eating tainted meat.

No microbe has yet been discovered, and there is as yet no evidence that the disease is insect borne, though analogy points very strongly towards this, and it must not be forgotten that it occurs in places and during the months which are specially beloved by many insects. The *Stegomya fasciata* (the transmitter of yellow fever) and the *Culex fatigans* are both common house mosquitoes in the towns affected, but whereas the *Stegomya* breeds in casual water in or near a house, such as that in tins, wash-tubs etc, and is not found in cess-pools, the *Culex* seems to prefer foul water like the contents of cess-pools, and cannot be bred in clean water in a laboratory. If, therefore, one of these two mosquitoes is to be suspected, the *Culex fatigans* is the more likely to be the transmitter. I should very much like to see the experiment tried in an infected port of killing off all the mosquitoes and seeing whether the disease would then not cease to be endemic, but it must always be remembered that there are other insects besides mosquitoes which are capable of transmitting disease, such as flies, by means of food to the alimentary canal.

One attack seems to confer immunity, and all observers agree

that the disease is not contagious, and therefore nurses do not contract it.

Varieties.—Besides ordinary cases of the disease, there may be very mild ones, or those which are dangerously severe. In mild cases, the symptoms may only be present for one week, there may even be no jaundice and there is no secondary fever.

The severe type is characterized by uræmia, cardiac paralysis, hyperpyrexia or hæmorrhages. The question as to whether it is right to group, as I have done, all cases of *infectious* jaundice with similar clinical symptoms under one heading can only be determined by future bacteriological discovery.

SYMPTOMS AND COURSE.

The *incubation* is very short, generally one or two days. The onset is sudden, mostly ushered in with a distinct rigor, and temperature between 39° and 40°, accompanied by pain in the muscles and lumbar region, headache and vomiting. These symptoms continue until the third or fourth day, when jaundice appears with marked enlargement and tenderness of the liver, enlargement of spleen and albuminuria, after which the fever usually subsides. The jaundice with the other symptoms gradually disappear, but in about three fourths of the cases the fever recurs for some days, and convalescence is in all cases very slow.

The important characteristics of the disease have caused writers to divide the symptoms into various stages, the simplest being (1) primary fever, (2) jaundice, (3) secondary fever.

The first stage lasts from 3 to 5 days, the jaundice from 7 to 9 days and the secondary fever again from 7 to 9 days, making a total duration of about three weeks.

In a typical case the fever usually remains between 39° and 40° for four days, and falls somewhat abruptly on the fifth day to between 37°.5 and 38°.5 where it remains stationary until about the ninth day, when there are some four days of normal temperature, followed by about a week of the secondary fever, which oscillates between 38° in the morning and 39° at night, after which convalescence slowly begins. In some fatal cases the

temperature rises to 41°.5 before death and has been found to be 42° or 43° in the rectum after death.

The pulse is quick during the fever, but may be as slow as 60 in the jaundice stage. This chart (fig. 1) is taken from one of Dr. Valassopoulo's cases in the Greek hospital, Alexandria.

The nervous symptoms are chiefly headache, giddiness, and sleeplessness, which usher in the fever and disappear with it. Delirium at night is not uncommon at the beginning, while bad cases develop somnolence, prostration, hiccough, weakness of sphincters, lividity of face, dry-brown tongue and muscular twitchings, in fact the "typhoid state." The muscular pains, especially at the nape of the neck and in the calves of the legs, are intense during the first stage of the disease, and are greatly increased by pressure, forming a useful diagnostic sign while one is in doubt about the nature of the illness.

Digestive Symptoms.—Patients generally complain of epigastric pain, which can usually be traced to the liver, but sometimes the pain is all over the abdomen. Nausea and bilious vomiting occur in the first stage, and constipation is the rule, but in some very severe cases diarrhœa takes its place. The stools, as might be expected, are of normal colour during the first stage and gradually become clay coloured and even white during the jaundice stage.

Jaundice is of course one of the most important symptoms. It usually begins on the fourth or fifth day, but in rare cases it may appear on the third, or not until the sixth or seventh day of the disease; it first appears in the conjunctiva (with bile pigment in the urine), and in a few hours spreads over the whole body, the shades being of various intensity. Some very slight cases may show little or no jaundice, and bile pigment cannot be found in the urine. In most mild cases jaundice disappears at the beginning of convalescence, but in some of the patients, who are stained almost a mahogany colour, the skin does not regain its normal tint for a month.

Hæmorrhages.—Epistaxis is the earliest and most frequent, it occurs at the end of the first stage, and may be seriously profuse. Intestinal hæmorrhage, during the second stage, is,

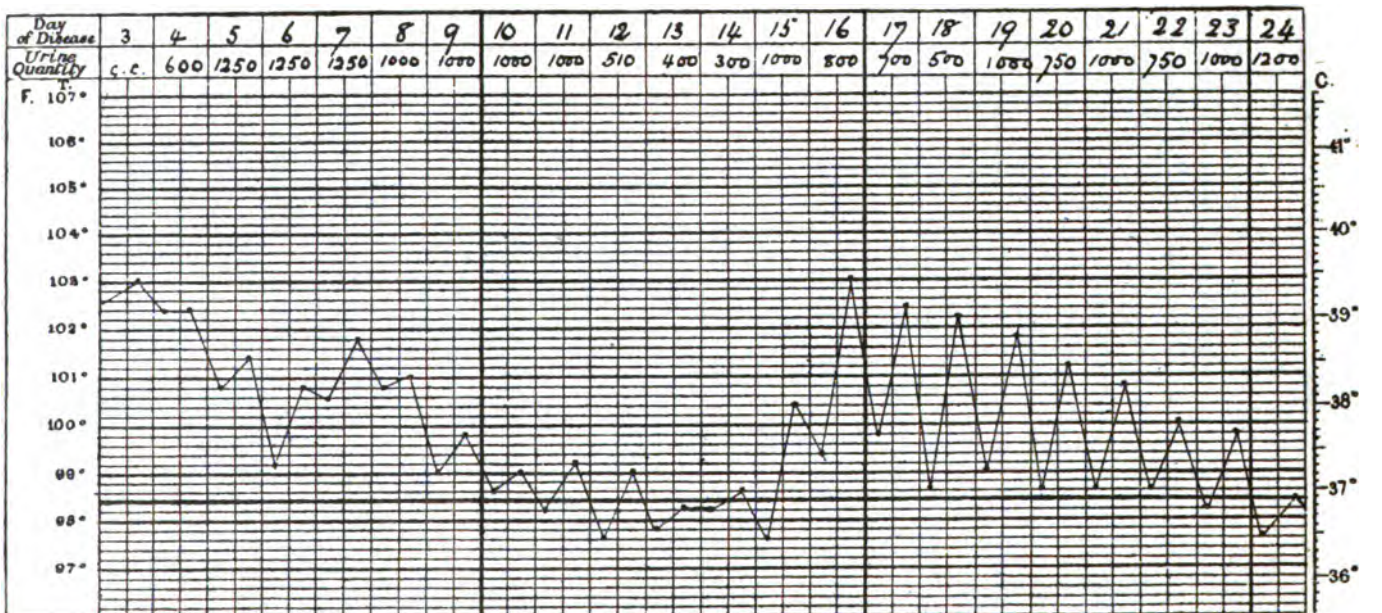
Day of Disease
3 4 5 6 7 8 9 10 11 12 13 14 15 16 17 18 19 20 21 22 23 24
Urine Quantity
c.c. 600 1250 1250 1250 1000 1000 1000 1000 510 400 300 1000 800 700 500 1000 750 1000 750 1000 1200
T.
F. 107°
106°
105°
104°
103°
102°
101°
100°
99°
98°
97°
C.
41°
40°
39°
38°
37°
36°

Fig. 1.

after epistaxis, the most likely to occur; hæmatemesis was only seen six times in 300 cases in Alexandria, but to these must be added occasional instances of melæna, which may have been of gastric origin. Hæmaturia is rare, but bloody sputa, hæmorrhage from the gums, and petechiæ on the skin are often seen.

Urine.—The changes in the urine in this disease are so marked that one sometimes wonders if the renal secretion does not contain the key to the mystery. During the first stage the urine, which is acid throughout, becomes scanty and of high colour, and albumen is nearly always present, but the quantity of urea is diminished. During the second stage, and more markedly in severe cases, all the above changes are accentuated. The urine diminishes in quantity till only 50 or 100 grammes are passed in the 24 hours, the colour becomes gradually greenish, bile pigment is seen, more albumen is present, and blood casts may be revealed by the microscope.

Now comes the urinary crisis between the 7th and 9th day, which is one of the most interesting phases of this remarkable disease, and which evidently betokens nature's effort to get rid of the poison. An excessive secretion rapidly takes the place of almost complete suppression, so that patients have been known to pass, on the second day of this crisis, urine varying in quantity from 2 to 8 litres in the 24 hours, while the specific gravity may be lowered from 1022 to 1005. The albumen becomes less, the quantity of urea is increased from perhaps 15 grammes to 50 in the day, and polyuria is continued even during the third stage of secondary fever. Leucin and tyrosin are usually absent, but they were present in one of my sporadic cases, which I half thought, during life, was one of acute yellow atrophy of the liver.

The *liver* and *spleen* are swollen and tender from the beginning, and can be felt below the ribs. Their relative vertical measurements by percussion reach to 7 inches and 5 inches respectively, but during convalescence they gradually regain their normal dimensions.

Mortality.—The official statistics until 1904 included this disease under a general heading of "fièvres typhiques" and

therefore it is impossible to get any figures from the general population. But Valassopoulo has analysed all the 300 cases admitted into the Greek hospital in Alexandria from 1875 to 1901, and found an average death rate of 32 per cent. The mortality of any one year may vary from 10 to 60 per cent, the highest figures occurring in small localized outbreaks. The death rate is highest in the spring at the beginning of an explosion, and lowest in summer when the epidemic is waning. But the epidemic at Nauplia which gave a mortality of 25 per cent on 187 cases, and lasted from June to December reached its highest death rate in July and August.

The disease is more fatal to stout than to thin people, and apparently to persons newly arrived in the country, and to those over 40 years of age. There was no death among 13 cases between 16 and 20 years of age in Alexandria, and 13 other cases at Nauplia, between 8 and 20 years, enjoyed a similar immunity.

As in other fevers, a patient may die at any time of some complication, or of debility caused by some co-existing disease, but ordinary fatal cases of this fever succumb between the 6th and 9th days from cardiac syncope or from uræmia.

Complications and Sequelæ.—Hyperpyrexia sometimes occurs at the beginning of the illness, the thermometer marking from 40° to 41°·5. It will easily be believed that this illness, which is nearly always severe, and is followed by a protracted convalescence urges the patient on the downward road of any disease to which he is already predisposed.

Pathology and Morbid Anatomy.—In the absence of evidence of inoculation through the skin, we must suppose that the virus is introduced into the body through the alimentary tract, after which the cells of the liver and kidneys suffer most injury, accounting for the jaundice and nephritis.

Many of the observers in Egypt have ineffectually tried to differentiate the special organism which produces the infection. Jaëger and Nauwerck in Germany discovered the "bacillus proteus fluorescens", which they believe to be the cause of Weil's disease, in the urine of patients, in their bodies after

death, and in ducks and geese which had died in the same locality of a fatal disease, the chief feature of which was jaundice.

Corpses usually show well marked 'rigor mortis', the skin and mucous membranes are jaundiced, and there are often petechiæ on the skin of the trunk and limbs. Petechial hæmorrhages, not unlike those of typhus, are variously distributed on the pericardium, endocardium, pleuræ, and peritoneum.

With the exception of hypostatic congestion, the lungs are apparently healthy.

The heart muscle is pale, soft and flabby, and the blood is liquid and dark in colour, as in other acute infections.

The liver is enlarged and shows signs of early fatty degeneration, the lobules are indistinct, the liver cells show cloudy swelling and the portal canals are enlarged and infiltrated with lymphocytes; some Egyptian cases examined in Paris for Valassopoulo were described as diffuse hepatitis with fatty degeneration.

The gall bladder is sometimes empty, sometimes full, it was distended to 150 grammes in one case; when full, pressure cannot squeeze out the thick bile though there is no visible obstruction.

Dr. Ruffer has found that sections of liver show emboli of liver cells in the veins, and he once found liver cells carried by the circulation into the kidney, also hæmorrhages in the liver and debris of the liver tissue and blood mixed up together. He has suggested that the liver changes are something like those produced by fat embolism after fracture of a leg.

The spleen varies much in size, it is always hypertrophied, but cases of great enlargement are probably due to chronic malaria; its average weight is about 500 grammes. The stomach shows capillary hæmorrhages in the mucous membrane, and there are sometimes ecchymoses in the ileum and colon. The kidneys also are the seat of similar hæmorrhages and are always large and congested, but the only microscopical change so far described is a slight infiltration of lymphocytes round the glomeruli.

There are no changes in the brain, except a general hyperæmia of the membranes.

Dr. W. Hunter has pointed out the similarity between the jaundice of Weil's disease, and that produced in dogs by toluylendiamin. This drug causes fever, swelling of liver and

spleen, changes in the kidney, bile ducts distended with bile and congestion of the duodenum. I append condensed notes of two autopsies on my cases made at Kasr-el-Ainy by Dr. Symmers.

Case 1.—A man about 24 years old, of a pale brown colour, was found in the street by the police, semi-comatose, but in a condition of slight cerebral irritation; his knee-jerks were exaggerated, and his limbs rigid, but there was no paralysis, there were no abnormal signs in the lungs or heart, the abdomen was somewhat distended and very tender about the epigastrium. He had retention of urine, constipation, and his tongue was dry, with sordes on his lips and teeth; he groaned continually, and had general jaundice, and his temperature was 37°·8, pulse 100. The liver seemed to be of normal size and did not extend below the ribs, but the spleen was much enlarged, and measured 17 c.m., of which 7 c.m. were below the ribs. Both liver and spleen were extremely tender when touched. The urine contained bile, and was acid, but there was no albumen. He died a few hours after admission, and the *p.m.* was made the following day. The whole of the skin and mucous membranes were intensely jaundiced, the body was that of a well-nourished man, and rigor mortis was disappearing in the lower extremities, the pupils were dilated and of equal size, and there were petechial hæmorrhages on the trunk, arms, thighs and legs. The pericardial sac contained 2 ounces of bile-stained serum, there was a considerable amount of bile-stained fat on the heart, and there were numerous petechial hæmorrhages on the epicardium; the heart was filled with large white clots, but the organs contained very fluid non-coagulated blood. The cardiac muscle was hypertrophied, the wall of the left ventricle measured from $^1/_2$ inch to 1 inch in thickness, and the heart weighed 410 grammes. The lungs were bile-stained, and there were a few petechiæ on the pleuræ, but otherwise normal. The whole peritoneum was much bile-stained, and there were numerous macular and petechial hæmorrhages on the parietal peritoneum. No ascites; the retro-peritoneal glands were very slightly swollen. The stomach was dilated, and much bile-stained, the lower part of the small intestine was very red, and apparently in a state of enteritis. The liver weighed 1950 grammes, and was very pale and jaundiced; the gall-bladder

contained thick bile. The spleen weighed 1030 grammes, being enormously hypertrophied and very firm and it contained a large number of yellow spots the size of pin heads. The right kidney weighed 230 and the left 240 grammes, both organs were very pale and contained a few petechial hæmorrhages under the capsules; the cut section was uniformly cream-coloured. but numerous pale pink Malpighian corpuscles could be seen. The other organs of the body were bile-stained but apparently normal. There were no bilharzia or ankylostoma worms.

Case 2.—A Sudanese servant aged about 45, was admitted to the hospital within 11 days of Case 1. He said that he had been ill six days, and if this be true he died on the 10th day of the disease. He complained most of jaundice, headache, constipation, tenderness over the liver and pain in the knees and upper extremities. His tongue was covered by a thick yellowish fur, his knee jerks were diminished. Urine 1010 sp. gr., yellow, acid, contained much bile pigment and albumen.

P. M. The body was well-nourished, the mucous membranes were extremely jaundiced, but the skin was too black to see jaundice or petechiæ, rigor mortis present. The heart was normal, except that the epicardium presented a large number of petechial hæmorrhages, especially on the anterior surface of the right ventricle. The lungs were normal, except for a few scattered petechiæ on the pleuræ. The peritoneum and external coating of the intestines were deeply jaundiced. The liver weighed 1950 grammes, and, though swollen, did not appear abnormal on section, the gall bladder was much distended with thick bile, which could not be squeezed out. The spleen weighed 257 grammes, the capsule showed numerous petechiæ, and in the pulp of the organ were several minute yellow spots. Each kidney weighed 225 grammes, and was swollen and very jaundiced, with numerous black petechiæ under the capsule, the cortex was very yellow from bile staining, and was also enlarged and fatty. The other organs were normal, but bile stained, and there were no entozoa found.

Diagnosis.—From the so-called bilious-remittent fever of hot countries, and from all other malarial fevers, this one can be diagnosed by the absence of Laveran's hæmatozoa. The disease,

in spite of its confusing nomenclature, has no resemblance with enteric fever or typhus, and some of the sufferers will be found to have already undergone an attack of one of these diseases. It must not be forgotten that in both enteric and typhus jaundice is a rare complication.

For many years infectious jaundice was confused with *relapsing fever*, but since the discovery (1872) of the *Spirocheta Obermeieri* the diagnosis is not difficult, for this organism is never present in infectious jaundice, though, if looked for at the proper time, it can invariably be found in the blood of relapsing fever patients. Both diseases are acute infections with jaundice, and both have recently occurred as epidemics in Alexandria, but it must be remembered that in relapsing fever the spleen is chiefly affected, while in infectious jaundice it is the liver which is enlarged, and suppression of urine and nephritis are also present.

Acute yellow atrophy is sometimes difficult to exclude in sporadic cases, though in that disease careful clinical examination can generally find that the liver is gradually diminishing in size, and leucin and tyrosin should be present in the urine.

Yellow fever has never been found in Egypt, but it has been suggested that the infectious jaundice of Alexandria is a modified form of this scourge. Some of the reasons for not accepting this view are that in yellow fever there is distinct albuminuria in severe cases on the first day of the disease, and in all cases except the slightest there is albumen on the second or third day, whereas in infectious jaundice the kidney symptoms come later, and the albumen is never excessive. The temperature charts do not betray any resemblance, and at *post mortems* the liver of infectious jaundice is not invariably yellow, as it is in yellow fever.

Are we justified in embracing under one heading the infectious jaundice which I have described from various parts of the Mediterranean and the cases called "Weil's disease" reported from Germany and other parts of Northern Europe? I think everyone allows that all the symptoms are very similar, and in many cases of Weil's disease there is a secondary fever, but some critics object quite reasonably that Weil's disease represents a milder illness with less mortality, and although sometimes of local epidemicity, it never becomes endemic: also that the spleen

is more enlarged and there is less fever than in infectious jaundice. Can these differences not be accounted for by variations in the climate, temperature, race and sanitary surroundings?

Prognosis.—The chief indications of danger are an irregular feeble pulse during the first week; early and intense nervous symptoms such as prostration, violent delirium or trembling of hands; extra deep jaundice colour; suppression of urea, much albuminuria, and the presence of blood and casts in the urine; multiple persistent hæmorrhages.

Total suppression of urine can be safely borne for a day or two, provided it is relieved by the urinary crisis.

Treatment.—At the beginning any intestinal antiseptics are worth trying.

The diet during the febrile period should consist only of milk with the addition of large quantities of water to drink. The patient must be kept rigidly in bed, and any heart symptoms must be met by injections of strychnia hypodermically. During the second stage, the quantity and quality of the urine must be carefully watched, the patient should be encouraged to drink as much water as possible, and two litres or more of artificial serum may be injected deeply under the skin to try and ward off uræmia. But if, in spite of all care, the patient is apparently dying of uræmia, venesection should be seriously considered, followed by transfusion of serum into a vein.

During the crisis, caused by the copious discharge of urine, our efforts to aid elimination are no longer necessary, but the patient will probably want extra stimulation instead.

Calomel and quinine are the drugs chiefly employed, but there is no evidence that either of them are of any avail, and the latter had better be reserved until the long convalescence, when it is very useful. (Valassopoulo.)

Prevention.—The little we know about this disease shows that it is produced, like so many others, by filth. In Germany attention has been drawn to the fact that butchers were chiefly affected, and various text-books have stated that it is chiefly butchers who suffer from Weil's disease, but the butchers in

question probably contracted the disease because they were specially exposed to contact with carcases of animals, or with untrapped drains. Soldiers and others have fallen victims by bathing in dirty water, those who work in drains seem peculiarly liable in certain towns, and though we know as yet little about this disease, it can safely be predicted that it need not be feared in any sanitary town. The drains of Alexandria, constructed before the English occupation by ignorant engineers, remain a disgrace to that prosperous city, and when they are thoroughly reformed and house sanitation is taken in hand infectious jaundice will probably disappear.

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SCARLET FEVER.

History.—The earliest record of this disease dates from the year 1543 in Sicily. It probably existed in Europe before that date but was confused with measles and erysipelas, until it was christened and differentiated by Sydenham, who described the disorder as it prevailed in London from 1661 to 1675. There is no evidence as to when or where it first reached Africa or Asia.

Pruner saw, in the winters of 1844-5, a few cases without deaths among Europeans, but never among natives; most of these were in Alexandria, but there were also a few in Cairo.

All observers agree that this disease was very rare in Egypt until 1887, but since that year very occasional cases, and even some rare deaths have been notified to the Sanitary Department, chiefly from Alexandria. The first case I ever saw was in June 1891, when an English child, aged eight, living in Cairo, had a typical mild attack. About the same time there were three cases among the children of English soldiers in the Kasr el Nil barracks, and it is possible that this infection was transmitted by post from England, where there was then an epidemic. In the summer of 1900, the English matron of Kasr el Ainy hospital caught the disease after attending a party at the Citadel hospital, where there were then several cases among the English soldiers' children, and in November of the same year 24 soldiers, quartered at the Citadel, suffered from it. The Citadel cases were originally probably due to clothing sent to one of the regiments from Scotland, where scarlet fever was prevalent. In December 1900, and again in January 1901 a case was diagnosed in the Cairo hospital for infectious diseases, and in April 1901 an American child, convalescing from enteric fever, travelled from Cairo to Marseilles, and was found to be covered with a well-marked scarlet fever rash on the day before reaching Marseilles.

The Egyptians call the disease "El homma el Kermizieh."

Distribution.—Scarlet fever is essentially a complaint of temperate climates, and in the tropics it is unknown as an indigenous disease, for when met with at rare intervals in India, China or Japan, it is invariably found affecting Europeans only. In subtropical countries the disease is rather more common, until a moderately cool climate is reached, when the disease becomes quite frequent both in the northern and southern hemispheres. Very extreme cold, however, seems to be almost as unfriendly to this fever as great heat.

It exists in all European countries if we exclude the most northerly, and in all of them except Russia, there has been a great reduction in the mortality of recent years.

It is said to occur occasionally among the pilgrims at Medina, and also in Mesopotamia and Teheran. It is very rare in Syria, Algeria, Tunis and Abyssinia, and is unknown in Central Africa, but in South Africa it is met with at rare intervals.

The only mention of it in the Sudan is by Hartmann, who says that it is called there "Burdjoq." There are many doctors who, after several years practice in Egypt, have never seen a single case. Personally I have never met with scarlet fever in a native, but Dr. Ibrahim Pasha Hassan has seen four cases in Cairo during the last 30 years, Mr. H. Milton tells me of three other cases and Dr. Comanos Pasha has met with it in two brothers. It seems certain that some races are more susceptible to the infection than others, for in the United States the disease is less prevalent and less fatal among the negroes and Red Indians than among the whites. In the fever wards of the German hospital in Cairo, where all nationalities are admitted, there have been of late years many cases of scarlet fever, but almost all of them have arisen among the children of the British soldiers.

Causes.—Egyptian experience bears out the general belief that children under ten years are specially liable. It seems difficult to understand why in Egypt the children of British parents should suffer more than those of other Europeans, unless the infection has usually been transmitted by post, in clothing, toys or dolls' garments. It used to be thought that the period of greatest infection corresponded with that of late desquamation,

but fever specialists in London now have a tendency to ignore desquamation after the fifth week, though they find that if rhinorrhœa or otorrhœa exist, cases may remain infectious for considerably more than 100 days.

Symptoms.—Europeans in Egypt take the disease in a very mild form, though the symptoms are sufficiently unmistakeable to be certain of the diagnosis; there are no marked swelling of glands, albuminuria, middle ear disease, or other complications and seldom a fatal result. An exception to this benign rule occurred in June 1903 when I saw, in consultation with Dr. Tribe, a young English banker, who died with hyperpyrexia, one of the rarest complications of this fever in any country. His temperature ran up from 38° to 40° and reached 42°·8 (109° F.) on the 8th day when he died.

The *Diagnosis* from tonsillitis may be difficult, but in the latter the simple erythema, when present, is usually limited to the neck and chest, and the tongue remains coated all over. In *measles* running from the eyes and nose are usually present, the fever falls suddenly within 2 days of the appearance of the eruption, instead of diminishing gradually, and the measles spots are first seen behind the ears. *Erysipelas* without an obvious wound is difficult sometimes to differentiate, but it is usually not accompanied by sore throat. *Dengue* is the only disease which I have seen confounded with scarlet fever in Egypt. In the autumn of 1887 several English soldiers were admitted to the Citadel hospital, suffering from a contagious fever, malaise and a red rash quickly followed by desquamation; this was not unnaturally called scarlet fever by doctors unacquainted with dengue. I was kindly allowed to see all the cases, and, though they seemed to be more like scarlet fever than any thing I had ever seen, I was at once struck with the absence in the whole group of enlarged glands in the neck, strawberry tongues and albuminuria. On the same day I saw similar cases in the town, and then became aware that some of them had come from Alexandria, where dengue was prevalent. Those of us to whom dengue was an unknown disease, then began to study it, and the diagnosis of scarlet fever was relinquished. It should not be forgotten

that dengue seldom occurs in Egypt except in the autumn. The rashes caused by belladonna and copaiba are scarlet, but are not accompanied by fever and sore throat.

Treatment.—Urotropin may be given during the third week if scarlatinal nephritis be feared.

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MEASLES

Synonyms.—Rubeola. *Fr.* Rougeole. *Ar.* Hasbeh.

History.—The Arabic writers, Rhazes (10th century), Ali Abbas, Ibn Sina and Ibn Zohr all refer to measles, though some of them seem to have regarded it as a modification of small-pox, and probably confounded it with the hæmorrhagic, abortive and other anomalous forms of that disease, for they considered it more dangerous than small-pox. Rhazes gives, however, the following symptoms: acute fever, hoarseness, redness of cheeks, pain in throat and chest, dryness of tongue, pain and heaviness of head, redness of eyes with a great flow of tears, nausea and anxiety—"when, therefore, you see these symptoms the measles are certainly about to appear." He states in several of his writings that a violet coloured rash (? hæmorrhagic) denotes a fatal attack.

It was not until the 17th century that measles was distinctly recognized in Europe from scarlet fever and small-pox.

Pruner says that measles in his time was mostly sporadic in Egypt, though there was an epidemic in 1845; he describes it as generally beginning in February or March, and reaching its height in May. He found among children from 7 to 18 months old a great tendency to complications, such as bronchitis, dysenteric diarrhœa and ophthalmia, all due to unfortunate domestic treatment, for the patients were kept 40 days on low diet, without fresh air, and those who escaped these complications ran a great risk of getting typhus.

Hartmann says that measles in 1864 was frequently in Egypt, and also in the Sudan, among children.

Distribution.—It is a disease affecting every race all over the world, but is always more prevalent in towns than in villages. It

now exists every year in Egypt, though not always in the same towns. It is the most fatal of all the infectious diseases among Egyptian children, for it causes one third of all the deaths under five years of age in the towns of Lower Egypt. This high mortality is due to the fact that the parents take little or no care of their children when ill, cases are never notified from the country villages, and the existence of the disease in any locality is only discovered by the Sanitary Department when attention is drawn to the increase of deaths from diarrhœa or broncho-pneumonia. The certified deaths from measles during 1902 were 644 out of 1589 cases. Very few cases are treated every year in the Government hospitals; the chief incidence of disease is between April and July. The soldiers in the Egyptian Army provide about three cases every year, and those in the Sudan about five cases. There is no great difference between the death rate of natives and Europeans in Egypt, but the figures for Alexandria are nearly three times higher than those for Cairo. There is said to have been a small epidemic at Dongola in 1904, and in Uganda reports show that measles is common, attended by broncho-pneumonia, caused by ignorance and insufficient nursing.

Complications.—Overcrowding, insufficient ventilation, bad nursing and scanty clothing are responsible for the diarrhœa and broncho-pneumonia which play such havoc with native children between the ages of six months and two years. All children under five years are more liable to these complications than older children. The broncho-pneumonia is not different from ordinary broncho-pneumonia, except that it forms with greater rapidity, and predisposes in many cases to tubercle.

Diagnosis.—Measles can be easily recognized even on black skins, for the papules stand out quite plainly, often in groups, and the coryza and Koplik's spots in the mouth are important aids. Scarlet fever and Rubella are very rare, so that Typhus is almost the only important disease likely to be confounded with measles in Egypt. In both fevers there may be injected eyes and a dusky face, but in typhus the rash is a kind of net work of small roundish spots on a dusky injection of skin, with-

out paler interspaces as in measles, where the eruption is in the form of crescents, horseshoes or straight lines. Moreover the measles rash appears on the fourth day behind the ears, while that of typhus is rarely well marked until the seventh day.

Koplik's spots can be found in almost every case before the rash appears.

Treatment.—The native customs are to wrap the patient up in a red sheet, or put his head in a long, red cap, I do not know the origin of this, but it is interesting to compare with the modern idea that red light is useful for some skin complaints. Forgetting the precepts of Rhazes, they never bathe the patient or even his eyes with water, (from a superstitious fear of ophthalmia) but they wash the eyes with the patient's urine, and give him black honey to drink. Honey was recommended by Rhazes and he also says "the stale urine of a man is useful in measles."

Not only should children, during the fever, be kept in bed in a darkened room away from healthy children, but cases of measles complicated with broncho-pneumonia should be isolated from those without this complication, because broncho-pneumonia is, apparently, a secondary infection, and by this system the death rate in some European hospitals has been greatly reduced. If an aperient be necessary, give only a small dose of castor oil, because of the special tendency to diarrhœa. Epistaxis, unless very severe, should not be interfered with, because it is usually beneficial. If it is impossible to get leave to sponge the child, fever may be treated by giving quinine in doses of 5 c.gm. three times a-day for each year of the patient's age, but with a maximum of 50 c.gm. per dose. Watchful care during convalescence is absolutely necessary to prevent lung troubles, and it is obvious that the cotton garments and bare legs of the poor are quite insufficient protection for the sick during the cold months. The child can be somewhat protected from chill by anointing the whole body with boracic lanoline.

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RUBELLA

I have never seen a case of this disease in Egypt, for it is certainly very rare, but I have been assured by one or two practititioners that it exists in Cairo sometimes, and that they have seen it in children who have already had an attack of measles. It is commonly seen among children between 5 and 15 years of age, but in England I have seen several cases over 30. It seems to be chiefly a malady of northern temperate climates, such as Germany, Great Britain and the Northern States of America.

SMALL POX.

Synonyms:—Small pox has been so named since the end of the 15th century to distinguish it from the pox, or syphilis; pox is the plural of pock, a bag or pustule. *Fr.* Petite vérole. *Ar.* Gidry (from the root, he sprouted); other names are mousim (the pilgrimage to Mecca, because it is usually an event that happens only once in a life time) and tarh, which also means sprouting.

Gidairy, or varioloid, is small pox modified by inoculation, vaccination or a previous attack.

Definition:—An acute, infectious disease, characterized by an eruption of papules, which, in the course of about eight days, pass through the stages of vesicle and pustule. Certain anomalous forms have more or less complete absence of the eruption, or hæmorrhages into the skin and from the mucous membranes.

History:—Small pox and inoculation for it are said to have been known in India and China since about 1200 B.C. Galen wrote of it, and it appeared in an epidemic form among the Abyssinian troops during the siege of Mecca in the Elephant war, about A.D. 570. But the earliest work on the subject is by Rhazes, who says that the malady was most prevalent in the latter end of autumn and the beginning of spring, or in winter if the weather was warm, and the wind southerly. He describes discrete and confluent rashes and says that black spots are followed by death. For treatment, he recommends bleeding from the basilic vein before the appearance of the eruption, copious draughts of very cold water to lower the fever, rose-water drops for the eyes, washing the face with cold water, gargles, many prescriptions of liniments to remove pock marks and liquid diet until convalescence. Ali Abbas and Ibn Sina

also wrote of small pox as being well known in the East, and it seems evident that the original foci of the disease were in India and Central Africa.

The doctors of Bonaparte's army considered the Sudan less infected than Egypt, for they noticed that slave caravans, arriving from Sennaar and Darfur, were attacked soon after they reached Cairo and this was the case, too, with white slave merchants. They found that more children in Cairo died of small pox than of plague; Pruner, on the other hand, forty years later, noted that small pox increased in frequency and severity as the Nile was ascended, until in the Sudan it appeared to be the one great sickness. In Egypt he found it chiefly between September and May, seldom confluent, usually attacking people under the age of thirty, and, like other writers, he pointed out the special predisposition of negroes to the disease. He only saw one case of the hæmorrhagic form in a large epidemic in Cairo in 1840—1. Fifty years ago the disease was still so prevalent in Cairo, that students in the Government schools often suffered from it, and 30 years ago most of the Circassian slaves in the hareems were marked by it. Hartmann says it was raging in Cairo and the Sudan in 1863—4; he gives some details about the native treatment in Kordofan, where patients were rubbed in sand, and healthy children too, to prevent their catching it, while in Darfur the sick were isolated.

Colucci Bey says that in August 1860, small pox attacked chiefly the negroes who had recently arrived in Egypt.

Of late years it has committed fearful ravages in the Sudan; in 1885, during the reign of the Mahdi, it is said that no less than 25,000 persons died of this disease in the town of Omdurman alone, and serious outbreaks occurred in 1891 and 1899. The Bishareen tribe is said to have lost one third of its number from small pox in 1889.

Distribution.—In 1903, which was an average year, there were 2,118 cases of small pox in all Egypt, of which 394 were fatal. The cases are scattered through all the various towns and provinces during each year, and are partly reinforced by infection arriving from neighbouring countries, where vaccination is not compulsory. For instance, in the Yemen district of Arabia,

small pox is rife, and among the pilgrims to Mecca, a very large number are attacked every year. At Omdurman it used to be endemic, and there was a small outbreak of it there in 1903, which was traced to importation from Tanta. The Egyptian army in Egypt during 10 years had only three cases, but the regiments in the Soudan, who are five times as numerous, had 466 cases, more than half of which occurred in 1900. The British army in Cairo and Alexandria has had small pox in 10 years out 15, the greatest number of cases being 42 in 1889.

I have seen a very few cases among English and American tourists, most of whom caught the disease in Upper Egypt. One nurse and a young surgeon, both English, contracted it while living at Kasr el Ainy.

A middle-aged Anglo-Indian once consulted me, in rude health, to know the name of the disease from which he had lately been suffering. He was covered with discrete scabs and desquamation, and stated he had travelled from Bombay in a steamer which had no doctor on board, and where he was the only passenger. He and the Captain had discussed the possibility of his having small pox, but dismissed this diagnosis, because he had had neither vomiting nor back-ache, and the only medical book at their command stated that these were necessary symptoms. It was before there were any infectious hospitals in Cairo, and he was living in a crowded hotel so that it was necessary to act quickly. I therefore put him in strict quarantine on a dahabia fastened to a secluded bank of the Nile. I chose a crew who had all had small pox, but, as a precaution, I vaccinated them all, though unsuccessfully. I then traced the progress of the steamer, and found that she was obliged to land small pox patients at every port at which she touched after leaving Port Said!

Causes.—All travellers agree that the blacks in the Sudan are more liable to this disease than the Egyptians or Turks, and that they get it in a worse form. Moreover, when the disease attacks a large hareem in Cairo, the negresses die, while the unvaccinated Circassians recover.

My own observation does not show that Nubians are less liable to small pox than the Sudanese, the following figures are taken from hospital patients:

	Adults examined.	Previous sm.pox.	Per cent.
Egyptians. . . .	324	69	21.3
Sudanese	59	55	93.2
Nubians	60	59	98.3

It is, however, rare to find any Egyptian under the age of 40 who has had small pox. Men over 50 caught the disease in their childhood, when vaccination was very little carried out by the Government.

I once examined a negro regiment which had never been vaccinated, and found that three fourths of the men bore evident small pox marks, the remaining fourth bore no certain marks, and some of them were in doubt as to whether they had had the disease or not, but I had them vaccinated, and almost everyone of them proved to be immune. This inclines one to the belief that almost all blacks of that class imported from the Sudan have had small pox there in childhood.

In the Sudan and Nubia the season of greatest prevalence is from the autumn to the spring, but in Egypt it is chiefly from March to June.

Since small pox hospitals have been established in Europe, it has been found that the virus can be carried by the air for a distance of 800 metres, and still retain its power of infection. This emphasizes the importance of always establishing hospitals for infectious diseases outside a town. Infection can be conveyed from the sick, or dead, from infected articles such as bedding, clothes, books, toys, coins or furniture, or from a healthy third person by means of his hair or clothing. The virus probably enters by the nose or mouth. The mildest form of the disease in one patient may produce hæmorrhagic small pox in another, or vice versâ.

A patient should not be allowed to leave hospital until the body, feet and hands are free from pustules and scabbing. If the pustules under the thick epidermis of the soles and palms, and under the nails, do not rupture, they must be cut out.

Special Symptoms.—The initial symptoms are as pronounced in mild as in severe cases, for the modification of the disease caused by vaccination does not appear until the eruptive stage.

It is useful, in diagnosing an early case, to remember that the *eruption* appears, as a rule, 48 hours after the onset of the illness. The earliest papules are usually found on the face, arms and back of wrists and hands, and later on the legs and trunk. They are not distributed uniformly, for the skin of the abdomen and chest, the flexor surfaces of the elbows and knees, the armpits and groins are but little affected. The eruption is most copious on regions of the skin which have been used to special pressure, such as that under the waist belt of the drawers, or under the garters. The papules vary in number from a few hundreds to several thousands. The disfigurement, helplessness, discomfort, and nauseous odour from a severe, confluent case, make this disease more trying both to patient and nurse than any other fever. It is wonderful that septicæmia does not more often occur.

The extent and depth of pitting left after convalescence depend on the amount of inflammation of the papillæ of the skin, and the consequent ulceration.

Since the days of Rhazes it has been known that special attention ought to be paid to the *eyes*, yet many deeply pitted Egyptians are blind of one or both eyes. This blindness is chiefly caused by the non inflammatory keratitis, which is apt to occur during the pustular stage in bad confluent cases. There are none of the usual premonitory signs of inflammation, the cornea quickly becomes cloudy, and may be quite opaque in one or two days, after which it sloughs, leading to general disorganization of the eyeball, or a portion only may slough, and the patient escapes with a staphyloma. Convalescence may vary from one week to several months.

Diagnosis.—Difficulty will arise both in the initial stage before eruption, and again after the eruption appears. Only those well acquainted with small pox can differentiate the early rashes, which may closely simulate scarlet fever or measles. The rash resembling *measles* is, however not preceded by coryza, is hardly raised above the skin, reaches its height within 24 hours of its first appearance, fades quickly, and is not accompanied by Koplik's spots.

We may suspect the onset of small pox, and therefore take

steps to vaccinate the household, if we find, in a feverish patient above the age of ten, a circumscribed, punctate eruption on the groins, sides of the trunk, lumbar region, or on the flexures of arms and legs, or an erythema affecting the external surface of the arms, hands, legs or feet.

The *typhus* eruption is not raised, and never appears before the fifth day of the disease.

In *influenza*, only severe cases should be mistaken, and then the prostration is more complete than in small pox, pains behind the eyes and in the limbs are usually complained of, and in cases of doubt the non-appearance of the papules on the third day of the disease will clear up the diagnosis.

Erythema multiforme, lichen, ptomaine and copaiba rashes and lumbago, in spite of the absence of fever, have all been mistaken for small pox.

But the most common errors occur at the beginning of the eruptive stage. The disease most often mistaken for small pox is *chicken pox*, and while I was in residence at a small pox hospital, we used to keep a ward specially for chicken pox cases sent there in error. In England, in 1904, chicken pox was temporarily made a disease which must be notified, in order that no doubtful cases of small pox should escape discovery.

Epidemics of very mild small pox may occur, and any large number of adult cases of a contagious disease with a pustular eruption probably betokens small pox. These are the chief differential points: (1) In chicken pox there are usually no initial symptoms, the first thing noted being the eruption on the trunk, limbs or face. If carefully observed, the spots are seen to begin as macules, which, in an hour or two, become papules, and in another few hours vesicles, practically all discrete. (2) The distribution of the eruption; for that of chicken pox is the opposite of that of small pox, being most abundant on the trunk, less so on the face, scalp, thighs and arms, and still less on the forearms, hands, legs and feet. (3) The unilocular character of the vesicles of chicken pox compared with the multilocular vesicles of small pox; in the latter the spots are hard, shotty, resistant to the finger pressed over them, and never burst, but in chicken pox all the full grown vesicles on the trunk collapse when the finger is drawn lightly over them. Chicken pox

vesicles on the extremities are less characteristic, being round, smaller and shotty-feeling, therefore the whole body must be examined before a diagnosis can be made. (4) The rate of growth of the vesicles; because in chicken pox they attain their full size in a few hours (8 to 24), and are then as large as small pox vesicles on the third or fourth day of the eruption, but in small pox the vesicles never reach their full size on the first day of the eruption. (5) The shape of the vesicle, for in chicken pox most are superficial and elongated or elliptical, later becoming dome shaped and distended with fluid, while in small pox the majority are circular and deeper. The unruptured chicken pox vesicle has no depression in its centre but the ruptured one often has, and then simulates small pox.

Measles has often been mistaken for confluent small pox on the first or second day of the eruption, but the finger can feel that the small pox rash is hard and shotty, while in measles, though slightly resistant, it is soft and velvety. The disease has been confounded with *cerebro spinal fever*, in which purpuric spots are not uncommon.

Syphilitic eruptions, herpes, impetigo, pemphigus, urticaria, acne, scabies, sudamina, pustular glanders and ulcerative endocarditis have all been diagnosed as small pox, chiefly because the observer had not examined the whole of the body. The effects of *mosquito punctures* are discrete, hard, shotty papules, but examination will show that they are limited to the exposed parts of the body. The effects are so similar at first sight to small pox that I have had to rescue more than one fair complexioned European from the unnecessary quarantine imposed after a colleague's visit. Lastly, the rashes due to iodides and bromides sometimes confuse hospital students.

Prognosis.—This depends chiefly on the previous vaccination, the mortality in unvaccinated cases at all ages is between 40 and 50 per cent, while in the vaccinated (without further revaccination) it varies from 3 at the age of 10 to 14, to 16 in adults over 50 years of age.

The number of marks also affects prognosis, for those with four or more good marks have a death rate four times smaller than those with one or two indifferent marks. A good mark

is one that shows a well foveated surface of more than one centimetre square.

The most favourable age is from 10 to 14, whether vaccinated or not.

If the initial symptoms are mild, the attack should not be fatal. Bad symptoms are: any form of hæmorrhage, general erythema with isolated black petechiæ, much eruption in the mouth and throat, and severe delirium. In bad cases of secondary fever the temperature may rise to 41°.5, and the patient dies between the 12th and 16th days. In hæmorrhagic small pox, besides the initial petechiæ and purple patches on the face and elsewhere, there may be bleeding from the gums, nose, stomach, lungs, bowels, kidneys or uterus and such patients usually die of heart syncope between the 3rd and 6th days, but the milder cases may occasionally recover.

Treatment.—A mild case wants little besides a bed and appropriate diet, but severe cases require most careful nursing, with plenty of air and free ventilation; they do well in tents or hospital huts, but if obliged to be treated in a house the patients should have a room measuring four metres every way to give him 64 cubic metres of air. Liquid diet must be given as in other fevers, but solids may be added when the appetite returns. Ice is very agreeable, and water should be given freely, for thirst is often excessive. The indications for stimulants are prostration and weak, rapid, compressible or intermittent pulse. The bed linen must be changed frequently, because of the pus which oozes in such quantities from the pustules, however comfortable the bed, the patient will feel as if he were sleeping on thorns. A hair mattress on wire makes the most comfortable bed, and a water bed is a luxury, though air and water cushions are almost useless. Boracic lotion must be applied constantly to the mouth, nose and eyes, while vaseline should be smeared on the eye-lids. It is very hard to keep the eyes free from discharge, because of the excessive chemosis of the lids. You must look out diligently for non inflammatory keratitis, and if it occurs apply warm poppy fomentations frequently, and drop atropine into the eyes twice a-day. A special attendant must be provided if the patient is delirious,

for small pox delirium is very violent; I have more than once seen a patient struggle with a nurse in order to try and escape naked from the hospital. Hypodermic morphia will quiet the delirium, and opium is the kindest remedy in all cases to gain rest, sleep and comfort. It is better to cut the hair short, because of the pustules and crusts from the scalp. Cold boracic compresses to the skin will allay the irritation, and antiphlogistin or boracic poultices will help the crusts to separate early. If a child or delirious patient is tearing off the epidermis, it is necessary to muffle the hands in bags. Abscesses must be specially looked out for, and opened when found. Laryngitis will require a steam tent, warm inhalations, and sometimes even tracheotomy. Convalescents from severe attacks will need tonics and cod liver oil.

Since 1893 Finsen has warmly advocated the red light treatment which he claims will prevent suppuration and disfiguring scars, but, unfortunately, recent experience in England and in Philadelphia has not corroborated this; the method is an old one, for it was employed in England 600 years ago. [1]

My own practice has been to paint the patients's bodies with a soft painter's brush about 5 centimetres wide using weak carbolic vaseline, but the most modern treatment is to apply pure liquid carbolic acid with a small camel's hair brush to the vesicles over a certain area of the body each day, beginning with the face and head, until the whole of the vesicles have been touched. Care must be taken to prevent the acid from running on to the healthy skin. This treatment prevents the offensive odour from the eruption, diminishes the chance of abscesses, relieves the irritation of skin and encourages the falling off of the scabs. If it is employed, carboluria must be watched for, though it seldom occurs.

The Egyptian treatment is to anoint the skin with salt butter or salt water, and apply almond oil to the eyes. The Berberins also rub a patient with ointment to assist the scabbing, but the important thing is to place him on a mat and bury all but his head in the sand for three days at a time, and squirt onion

[1] Gilbert was the first writer to suggest this and John of Gaddesden wrapped a royal prince in scarlet cloth and surrounded him with red hangings, and as a result there were no scars.

juice into his eyes to preserve his sight. The Sudanese regard small pox as a fetish, and, unlike other diseases, they make no attempt to treat it, they thrust the sick outside the village, and if the disease still continues to spread they abandon their homes to the sick, and start a new dwelling place one or two days journey from it.

Prevention.—There is only one sovereign remedy: vaccination during the first six months of life, and revaccination at the age of puberty. This should be compulsory, and will banish small pox, as has already been proved in Germany. After the initial symptoms of small pox have appeared it is quite useless to vaccinate the patient, but all those in his house should be revaccinated at once. If revaccination is done within three days of the reception of infection, and if it be successful, small pox will not occur, because the incubation period of vaccinia is shorter than that of small pox. If done later, it may not modify the attack. The sick man must be isolated, and, if possible, removed to hospital, the clothing must be burnt, and the room and all its contents sterilized. Dead bodies should be washed with 1/1000 sublimate lotion. Doctors and nurses exposed to infection should be revaccinated every two or three years.

It must be remembered that small pox may occasionally occur in an adult, even after revaccination at puberty, also, that in times of danger no age is too early for vaccination, and no state of health, except the presence of serious, acute disease, need contraindicate it.

INOCULATION AND VACCINATION.

The Chinese have made use of inoculation since the 11th century, powdered scabs of mild cases of small pox being still rubbed into the nostrils or the region of the umbilicus of children between the ages of three and six. For some centuries inoculation was practised on the forearm in India and also in Arabia. In Georgia and Circassia, since at least 1712, the method employed was for a woman to prick with three pins the precordial region, the epigastrium, the umbilicus, the palm of

the right hand and the left ankle, while in Barbary incisions were made on the back of the hand between the index finger and thumb. In Greece inoculation is said to have been common since 1537, the marks being made in the form of a cross on the forehead, cheeks and chin; this mode of treatment was carried from the Morea to Constantinople, and thence to England. Inoculation has been illegal in England since 1840. The most common method of inoculation in Egypt and the Sudan was to collect small pox matter on a rag and rub it into the scarified skin of the arm or forearm, or of the back of the left hand between the thumb and index finger, and to repeat the operation on the third day. If this was followed by fever, the inoculation was considered successful.

Vaccination is called by the Egyptians "tateem" or "tilqyha," (fecundation), being the word used for the artificial fecundation of the palm tree by introducing the pollen of the male into the female. The common people further call it "duqeh," or tattooing. The Arabic words used for varicella are also employed to denote any sequelæ which may follow careless or unclean vaccination.

The operation was introduced into Egypt by Clot Bey in 1827, by means of 2500 barbers, who had to be trained for the work. A central vaccination station was made at the Ezbekia hospital in Cairo, where 15,000 children were annually protected from small pox. According to Clot Bey, some 60,000 children used to die of small pox every year in Egypt before his time. Hartmann says that after this time the native doctors used to falsify the vaccination returns, and one of them in 1848 was condemned to the galleys for doing so. He also states that slaves, as they were being brought down the Nile, were vaccinated at Assuan, Kena and Assiut. The horrors of small pox as a general scourge are still well within the memory of old men, and therefore the Mussulmans have great faith in vaccination, though some of the most ignorant still believe that small pox is an advantage in that the eruption evidently purifies the blood of disorders. The intelligent ask that clean lymph be used, and some are averse to submitting babies of tender age to the vaccinator. The Copts have their children done on the 40th day after birth in three places on one arm, and if the operation is not successful, they repeat it on the other arm.

The Jews vaccinate healthy infants at the age of two months with six vesicles, three on each arm, and if the operation is not successful, it is repeated two or three times, for they have great faith in it, and small pox is consequently rare among them. In my enquiries on the subject I found that it was only they who seemed aware of the fact that syphilis could be communicated by human lymph.

The Bedawin near Cairo firmly believe in vaccination and triumphantly point out that now-a-days very few of them are marked by small pox, but their brethren in the Behera, Fayûm and Upper Egypt are further removed from civilization, so some "wise man" of the tribe either practises inoculation direct from a small pox patient, or takes vaccine matter from a cow, and if he has no knife or razor, he uses a sharpened ostrich feather to scratch the patient's skin. The Bedawin are very much afraid of small pox, and rigorously avoid all contact with infected villages.

The Berberins, on the other hand, are hardly afraid of it, believing that it is a necessary evil which it is well to get over young before others are dependent on them, moreover, they hold that the mortality among children is not so high as among adults. They therefore expose healthy children to infection, and, if this fails, they inoculate by a method which they probably learned long ago from their Turkish ancestors. A barber scarifies with a razor the skin of the arm or chest, and then, dipping a piece of cotton wool into a small pox pustule, he applies it to the wound. In the outlying parts of the Sudan, which vaccination has not yet reached, inoculation in the leg or arm is still practised.

These native customs are worth recording, because they are gradually disappearing before the spread of vaccination by the Government.

In 1883 vaccination was only carried out by Government doctors in the 14 principal towns of Egypt, 28,496 successful vaccinations were done in that year, representing 72 per cent of the town births, but all the country districts were left to their own devices. In 1903, 406,856 successful vaccinations were performed in Egypt, but only 11,000 in the Sudan. But it was not only the number of vaccinations which I found at fault in 1884, the

method also of performing the operation was highly dangerous. The public vaccinators of Cairo and Alexandria used to operate with a lancet on three places on each arm; on the eighth day they scraped off the scab, and vaccinated babies with the blood and lymph from the open sore. They then made a reserve supply by cutting up the scab into small pieces, and mixing it with blood and lymph, and this was preserved between two thick squares of glass. No care was taken to avoid sepsis, no inspection was made to see if the vaccinifer were healthy, and under the circumstances it was perhaps well that the medical students had no opportunity of learning how to vaccinate. I saw cases of ulcerating sores, general erysipelas, papular and pustular eruptions, and even death after the operation. In 1886 I further saw a chancroid ulcer on the arm, followed by a syphilitic eruption in an out-patient, and on another occasion a child of two years, with a history of a chancre on the vaccinated arm at the age of two months, syphilitic eruption and mucous patches on the lips and throat; the mother of this child had then suffered from infection near the nipple, sore throat and general eruption, which caused her to be divorced by her husband. The late Duruy Bey also told me that during 30 years he had seen ten native children who had been infected with syphilis at the time of vaccination. I urged upon the Government the necessity of introducing calf lymph for all Egypt, and after many initial difficulties this was eventually done. The Vaccine Institute, attached to the headquarters of the Sanitary department in Cairo for the supply of all Egypt, is now excellently organized under the control of the veterinary surgeons, and the students of the medical school regularly attend it before they are allowed to vaccinate the foundling children attached to Kasr el Ainy. In spite of mixing the calf lymph with glycerine and the use of centrifugalizing and ice machines, there are always difficulties in hot weather, which have so far prevented efficient vaccination in the distant parts of the Sudan. Weaned buffalo calves, aged from 4 to 6 months, are used in Cairo, because they are cheaper, larger, and have an almost complete immunity from tubercle. All calves are, however, previously subjected to the tuberculin test, and their organs are examined after death before the lymph is served out.

The country barbers are brought up to Cairo from time to time to learn the art, and when they return to their villages they receive P. T. 1 for each successful vaccination, and their work is superintended by travelling inspectors. This system is now being gradually extended to the Sudan.

Europeans who come from countries where revaccination is regularly practised escape small pox, and only a few careless people contract it. A striking example of this was furnished in 1896 in the Italian campaign in Abyssinia, where the revaccinated Italian troops at Massowa had not a single case, though small pox was raging among the unvaccinated native population. American visitors are fully alive to the importance of revaccination, but many of them object to more than one insertion, on the ground that their physicians at home consider this sufficient. Theoretically this should be true, but as a matter of fact, all statistics show that four or five marks are necessary to ensure adequate protection.

There are various ways of performing vaccination, but this is the method which I consider the best: When once opened, the contents of the tube must be used at once. The operator must have surgically clean hands and a sterile lancet, and the arm or leg of the patient must be washed with cotton wool and boracic lotion and then dried. The vaccine tube should be cut with scissors, or broken at both ends, and a little of the lymph allowed to drop in five places about 2 centimetres distant from each other. The point of the lancet should be gently scratched through the lymph until blood is seen. The operation is then completed, but, after waiting half a minute, I usually make certain with the flat of the lancet point that the blood and lymph have coalesced. A skilful vaccinator draws almost no blood, and can operate on a sleeping child without waking it. It is convenient to vaccinate infants while they are feeding from the breast or bottle, after which they often sleep. One of the essentials to secure success is to allow the scarifications to dry naturally, which takes at least half an hour, and during this time flies must be prevented from settling on the wounds. When the vesicle has formed, the best local treatment is boracic lanoline thickly smeared on lint, which allays irritation, keeps the wounds clean and prevents their sticking to the clothing.

Aseptic precautions and rest must be continued until all the wounds have dried up. By this method all five insertions are successful, and there is no risk of abscess, erysipelas, septicæmia or syphilis. Local troubles, such as erythema, sudamina or eczema should give only temporary discomfort. I have never met with an unvaccinated person who was unsusceptible.

Primary Vaccinia, or cow pox is a real disease, and should be treated seriously in children and in adults. The operation of vaccination is so simple and so universal that the constitutional symptoms are often neglected. The fever is present from the 4th to the 9th day, and may rise as high as 40° though it is seldom above 39°. Restlessness, crying, thirst, want of appetite, diarrhœa with green motions and loss of weight are the chief symptoms in infants. This chart (fig. 2) is an average one, taken from a healthy child aged six months.

Human small pox lymph inoculated directly on calves, almost invariably gives negative results, but Dr. Copeman has now succeeded in deriving vaccinia from variola after passage through monkeys and calves. This proves the long suspected identity of the virus of small pox with that of vaccinia, and confirms the view that the cow pox of Jenner's time was derived from a mild form of human small pox, such as that following inoculation.

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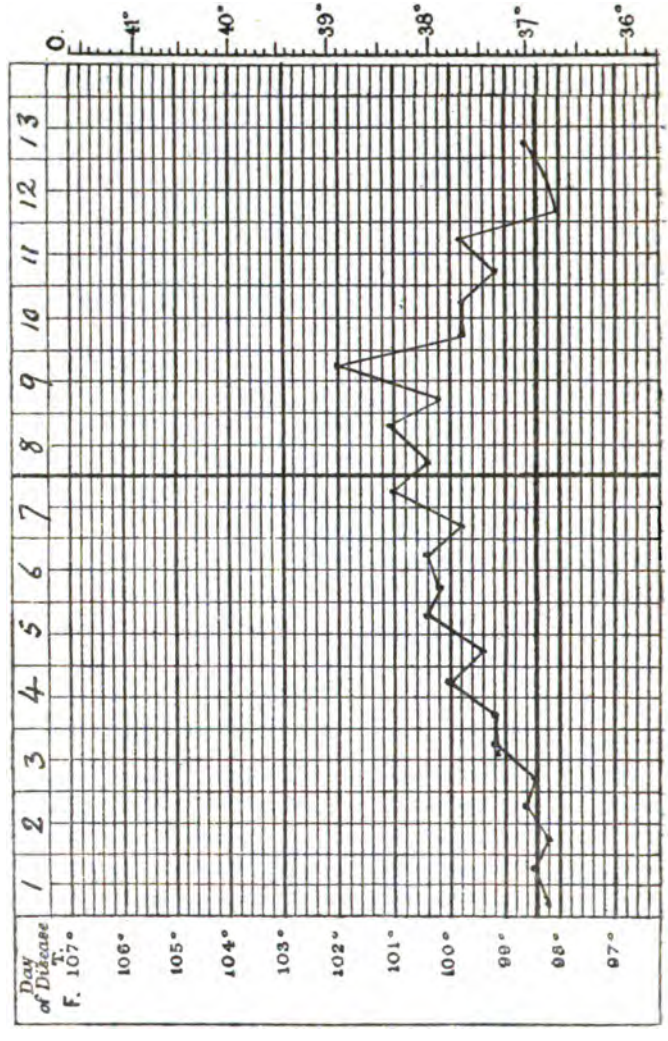

Fig. 2.

CHICKEN POX

Synonyms:—The English name is derived from the resemblance of the eruption to chick peas. *Ar.* Gidry kazab (false small pox), gidry el firakh (chicken's small pox) and gidry el homar (donkey's small pox). In the Sudan it is sometimes called borgool.

History.—Rhazes and Ibn Sina saw varicella as well as variola, but the two diseases were not identified till 1767. Since then it has been recognized that chicken pox is quite distinct from small pox, varioloid and vaccinia, though errors of diagnosis still occur in all countries. Hartmann says that varicella is very common in the Sudan, and Burton relates that in Somali Land children suffering from it are bathed in sheep's blood, after which they are wrapped up in a skin and seated in the sun.

Distribution.—It is an endemic disease in most parts of the world, occasionally becoming epidemic. There is distinctly less in Egypt than in Europe, and it is responsible for few or no deaths in the year. The natives probably always regard it as a mild form of small pox, but occasionally cases are brought to the hospitals for diagnosis. In the Sudan it is still quite common, and provided 431 cases during 10 years in the Egyptian Army, the greatest number being 126 in 1902. During the same year there was only one case in the Egyptian Army serving in Egypt. I have seen several cases in Cairo among adult negresses.

This chart (fig. 3) is from a child aged nine. One attack confers immunity, and each case is infective from the beginning of the eruption till the end of desquamation, or for a period varying from two to four weeks. The skin may remain red during all

this time, and any deep ulceration will be followed by pitting, which, in after life, will closely resemble small pox marks.

The *diagnosis* from small pox has been fully detailed. Cases of acne and syphilis should not cause confusion.

It is well to keep the patient in bed during the febrile stage.

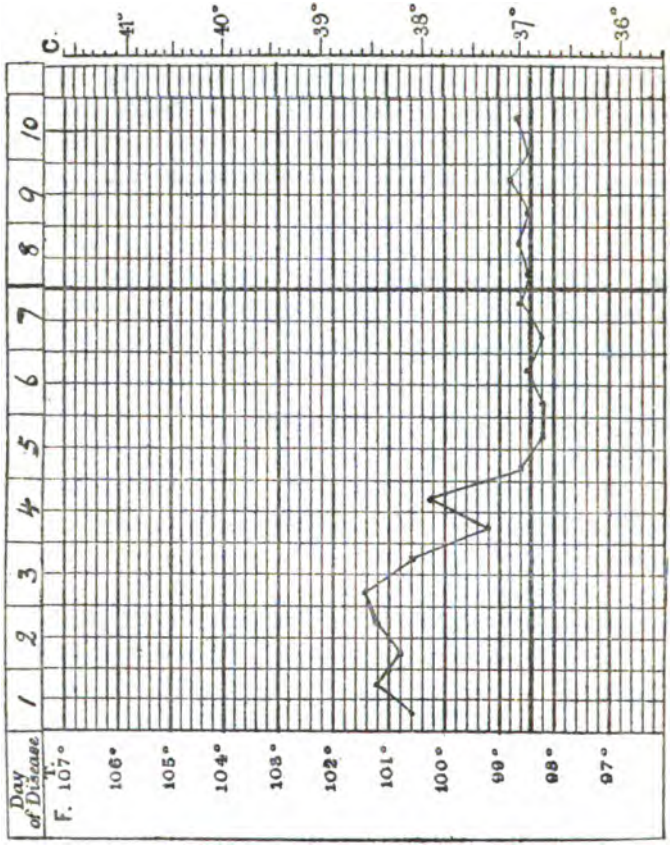

Fig. 3.

MUMPS

Synonyms:—Fr. Oreillons. *Ar.* Iltihab el ghoda el nukfieh.

History.—Mumps has been known since the days of Hippocrates, who himself observed, during an epidemic at Thasos, that orchitis was an occasional complication. Pruner saw a few cases among young people in Egypt, sometimes with orchitis.

Distribution.—This disease has been reported from all parts of the world. There are sometimes small and very mild epidemics of it in Egypt during the winter, and it has always seemed to me less contagious than in England. At the beginning of 1903, eight boys in the Military school of Toura suffered from it. The Egyptian Army statistics show cases every year from 1892 to 1902, the maximum number being in 1895, when there were 92 cases in Egypt and 58 in the Sudan.

The only complication I have ever seen is orchitis; this occurred in two young men a few days after the parotitis had subsided, both testicles were involved in each case, and with both of them marriage has since been sterile.

WHOOPING COUGH

Synonyms:—Fr. Coqueluche. *Ar.* Sowal deeky (crowing cough), Kokhet el deek.

History.—The first certain record of whooping cough was an epidemic in Paris in 1578, and for the last two centuries there have been numerous accounts of epidemics all over the world.

Distribution.—No country is free from it, but the prevalence and mortality are both most marked in cold regions during damp cold weather.

Very few cases are brought into the hospitals in Egypt, but the disease is endemic in the large towns, and sometimes becomes epidemic, with or without measles. The chief mortality is in Egyptian children during the first year of life, but the statistics show fewer deaths now than they did before 1890.

The children of tourists have sometimes brought whooping cough with them to Egypt, but I never saw any outbreak among the European residents until the winter of 1903—4, when a child arrived from Europe suffering from it, and infected a number of children at a party. Some of the mothers also contracted the disease, and although some of the cases were complicated with pneumonia, the attacks were milder than synchronous ones in England, none of the patients being dangerously ill. It must not be assumed that adult Egyptians will have undergone the disease in early life. In Alexandria it is more common than in Cairo among the Europeans. In Suez there are cases every year chiefly among the native children, many of whom die from broncho-pneumonia.

Symptoms.—Vomiting is a very serious complication in severe and protracted cases, because it tends to weaken the patient.

Very young children often refuse food for days, and then require very careful nursing, and perhaps feeding every half hour. The whoop generally begins after a few days, and the younger the child the earlier it occurs.

The *diagnosis* is often delayed until the whoop is first heard, but suspicion should be aroused if one meets with a patient attacked by sudden onsets of coughing, without any physical signs in the chest to account for them. The increased cough at night and vomiting after coughing should also help the diagnosis. It must be remembered that an adult may have the disease for weeks without whooping.

Treatment.— Parents should be warned of the serious nature of this disease, and prevented from allowing their infected children to attend schools or mix with healthy children. There is no certainty when the infection ceases, but quarantine need not be continued longer than six weeks from the onset of the illness, even if the patient is still whooping, for this may remain as a "habit." The sick child should be kept in a uniform temperature of 20° C. day and night, because the mucous membrane is, from the onset, in a state of catarrh, but on warm days children in Cairo may be out of doors for an hour. A bad case should not be thoroughly washed, or bathed at all, because this invariably brings on a convulsive attack.

All food difficult to digest increases the cough and vomiting, peptonized milk is useful, and, in severe cases of vomiting, it is better to feed the child immediately after an attack.

An enormous number of drugs and domestic remedies are employed, most of them being quite useless. I have given a French remedy, "Sirop Lagnoux," containing valerianate of caffeine, with some success; it reduces the number of paroxysms and the tendency to vomiting.

As soon as the patient is fit to be moved a change to desert air acts like magic.

The only *popular* treatment worth recording is that the unfortunate patient is made to drink some of his own urine.

GLANDULAR FEVER

This disease was first described by Pfeiffer in 1889, but it is still neglected by many authors. Since 1894 I have seen several sporadic cases among English and Egyptian children in Cairo, and I believe it is one of the predisposing causes which leads to the chronic enlargement of cervical glands in many young Egyptians. It is often associated with adenoid growths in the naso-pharynx.

The absence of pharyngitis and tonsillitis as symptoms, confirm the impression that the infection is derived from the intestines, and not from the pharynx or tonsils. The highest temperature I have so far seen is 39°.4, both morning and evening on the third day. No rash is present. The convalescence lasts one or two months, during which time the child is anæmic, and may have slight diarrhœa, and the protracted recovery may be the first reason for supposing that the original illness was more than an ordinary "cold in the neck". The diagnosis may have to be made from mumps and tubercular cervical adenitis.

INFLUENZA.

Synonyms.—This name was first used by an Italian writer in 1580, to mark the influence which the disease had in general diffusion over Europe, Africa and the East. The French call it "la grippe", the Americans "grip", and the Egyptians "aiya el moda" (the fashionable sickness), but bad cases are veiled under "naushah".

Definition.—An acute infectious disease, occurring in pandemics at irregular intervals, and then remaining in the form of recurring epidemics; it is characterized by toxic invasion of one or more systems, of which the respiratory is the most often attacked.

History.—The earliest record dates from an epidemic about 412 B.C. mentioned by Hippocrates, and this is followed by several similar accounts at long intervals, but, between 1173 and 1875, Hirsch records 85 outbreaks, of which at least 24 were pandemics.

The first notices I can find of influenza in Egypt were in March 1833, in January 1837 and in the spring of 1842, according to Pruner. He says the illness lasted two or three weeks, the Syrian troops suffered much, and some cases were complicated by pleurisy with effusion, but it was only dangerous to those with pre-existing lung disease. In 1860 there were some gastro-intestinal cases in Alexandria.

There are several points about influenza epidemics which distinguish them from all others. No other disease spreads so rapidly, attacks simultaneously so large a number of persons exposed to it, nor extends so universally over the world.

In 1874—5 we know from various writers that it existed in many countries of Europe and in the United States, but after those years nothing more was heard of it until 1889 though it

now seems evident that influenza, in an endemic, smouldering form, existed about 1888 in India, China, Russia and elsewhere. For some unknown reason the disease broke out with great virulence in Western Siberia in September 1889, and so rapidly invaded the world that within three or four months nearly all Europe, the United States, Canada, Mexico, Egypt, India, Persia and South Africa had all been affected.

Since that disastrous winter almost every habitable spot on the earth's surface has suffered from recurring epidemics of influenza, but in no instance has the infection travelled faster than an ordinary passenger could by postal service.

In 1892 Pfeiffer and Kitasato both discovered the bacillus which is the cause of the disease, and put an end to the fanciful theories that influenza was conveyed by some mysterious winds of the air.

Distribution.—I have only treated an average of ten cases every year among in-patients at Kasr el Ainy, mostly among policemen, which is an illustration of the fact that Egyptians do not enter the hospital for what they consider mild diseases.

The total number of deaths returned as influenza during the nine years 1892 to 1900, was, for the towns of Lower Egypt, 114 Europeans and 700 natives, and from Upper Egypt 269 natives, but to these figures must, of course, be added many deaths returned as broncho-pneumonia and other complications.

In 1889 the disease reached Cairo in November, spread to Alexandria and the rest of Egypt in December and January, and disappeared for a time in April and May 1890. It reached Suakin in February, and in March was found at Assuan and Wady Halfa, and probably attacked the Sudan, which was then under the Mahdi's rule. Most of the schools of Egypt had to be closed in February 1890, and again in 1892. In the former year 30 per cent of the scholars were attacked, besides teachers and servants, but not a single death occurred. More than half the doctors became victims, and at least 10 per cent of the English and Egyptian armies suffered, while it is fair to calculate that about one third of the whole population were attacked. In the winter of 1890—91 we saw several cases which had been introduced from Europe, but there was no general epidemic. Many of the residents, however, caught the influenza, and I,

personally, in my second attack, was ill from the effects of it for six months. In November 1891 the malady was again brought to Egypt by people returning from Europe, and spread all over the country between December and April, though it did not reach Suakin till July. It was quite as serious in its nature as the first epidemic, and attacked, again, at least one third of the people, though several of them had previously suffered from it. In the towns, 442 deaths were directly attributed to it during the first three months of 1892, and in February of that year so many of the Egyptian troops succumbed, that the military manœuvres at Assuan had to be abandoned. Yet, curiously enough, the winter visitors at Luxor escaped the disease, perhaps because recent attacks had rendered them immune. Since that time influenza has been imported into Egypt regularly every year, generally by visitors from Europe, but sometimes by passengers from the East. More than once a steamer has landed in the Suez Canal a score of influenzic patients in every stage of the illness, and these have told us that they have left many more behind on the vessel. This number of patients on the high seas is partly due to the fact that influenza is not considered a contagious disease which demands disinfection, and, partly, that whole families are escaping from the cold of Europe to bask in a southern climate, and these start as soon as one member of the party is convalescent from influenza; but no sooner are they at sea than another falls ill, a third succumbs a few days later, and this last case reaches Egypt in time to infect waiters and others in the hotels. The whole series of annual ebb and flow of influenza outbreaks in Egypt is a very interesting proof of the human transmission of the disease.

The sick rate in the British troops has varied from no cases at all returned during three years (1891, 1893, 1897) to 425 sick in 1900, and 361 in 1901. The Egyptian Army escaped without a reported case in the year 1897 only, whilst in 1900 there were 127 returns from influenza in Egypt, and 89 among the soldiers in the Sudan.

Causes.—Every age, sex, nationality and occupation is affected, but infants take the disease so mildly that it may escape detection if no complication occurs. I have had several patients over 75

years of age, who have successfully withstood an attack. The greatest number of sick come from the ages of 20 to 40. During some winters epidemics lasting some weeks have been confined to the Europeans, and at other times natives only have suffered, but this is only due to lack of free intercourse. Doctors and nurses are specially liable to contract this disease from their patients. No climate is inimical to influenza, which, when once it starts in an epidemic form, will spread anywhere, but it prefers cold, damp weather, and is, therefore, never such a serious disease in Egypt as in northern Europe. Its favourite months in Egypt are January and February, after which it is usually banished by the warmer, drier weather, or by the increased facilities for leading an outdoor life.

Contagion.—There have been innumerable instances of late years to prove that the disease can be easily acquired from patients with fever or cough, but both medical men and the general public are very careless about spreading the infection. Those predisposed to the disease have learnt by experience the folly of visiting friends ill with influenza; kissing, or being coughed upon are even more dangerous, and a wise traveller, during an epidemic, will try and make certain that his railway sleeping berth, steamer cabin and hotel mosquito net have not been inhabited by an infectious predecessor. In addition to these common causes, I have seen patients infected by a dentist in the acute stage, and by table waiters who breathe heavily into their faces while handing them dishes. During some of the Cairo epidemics, when the hotels have been crowded, the waiters have suffered in great numbers, and the least ill have had to continue their work, while the severer cases were sent to the hospital. As in other countries, it has been noticed that the insane are, apparently, somewhat less susceptible to influenza than the sane.

It should now be fully recognized that a patient is capable of conveying infection from the time of the onset of the earliest symptoms for at least a week, or until all cough and expectoration due to influenza are at an end.

A few persons appear not to be liable to the disease, but others are extraordinarily susceptible to it, and I have seen several who have contracted it at least a dozen times since 1889.

In spite of some clinical similarity there is no proof that the disease in horses is identical with that in man.

Symptoms.—The incubation period varies from one day to five, and is usually two or three days. No disease presents such a rich variety of symptoms and complications, and it therefore becomes extremely difficult to foretell in which way a given individual will suffer. The earliest victims in Egypt in 1889 became ill from the catarrhal or respiratory form, the prudent took to their beds and were well in a few days, the foolish or ignorant continued their usual avocations by day and night, and suffered from broncho-pneumonia for two or more weeks. The following winter we saw members of a household suffering from complications affecting one of various systems, respiratory, digestive or nervous, and it almost seemed as if the bacillus had a power of discrimination. Those with delicacy of the respiratory organs were attacked in their breathing apparatus, or slumbering tubercle bacilli were called into action. Vomiting, diarrhœa and colic were the lot of the dyspeptic, while the brain worker suffered in his nervous system with functional and even organic troubles.

The influenza poison probably always enters the body by the respiratory mucous membrane, generally developing there, but occasionally it passes on to the stomach or nervous system to develop in them, without affecting the breathing organs.

In rare cases the poison concentrates itself chiefly upon the heart, causing syncope, a feeble, irregular and rapid pulse and a tendency to sweating. Mental depression, which is so extreme that many men, with minds well balanced until this scourge, have seriously contemplated the idea of suicide, or have entreated their medical attendants to poison them, headache (frontal or orbital), inertia, sleeplessness, irritability, prostration and neuralgia are among the common nervous symptoms.

The tongue, in severe cases, is tremulous, large, soft, moist, thickly coated, and indented by the teeth, and this nauseous symptom, with loathing for food, may continue throughout convalescence, while, if the patient is forced by nurses to eat, diarrhœa and digestive troubles are sure to occur.

The cough is peculiar in being hard, dry and apparently unnecessary, for it does not ease the patient's chest, and it

greatly aggravates the headache. It is sometimes paroxysmal and does not yield to drugs, which only upset the already delicate stomach. The broncho-pneumonia is like that more usually met with in children, and has its chief focus in adults about the middle of the lungs.

Though fever is nearly always present at the onset of the illness, and again during bronchial complications, I have seen a few bad cases of the gastro-intestinal and nervous varieties, which never had a rise of temperature, though this was regularly tested.

Speaking generally, the natives of Egypt are much less liable to the nervous forms of the disease than are Europeans.

Of true relapses I have seen very few, but of unexpected complications, causing a renewal of fever, a great number.

After effects.—Almost every organ and tissue in the body has become impaired as the indirect result of influenza. Hair prematurely whitened, asthenopia, rapid decay of teeth, anæmia, emaciation, asthma, chronic laryngitis or tonsillitis, pneumonia, tubercular lung disease, tachycardia, nephritis, diabetes, loss of memory, and peripheral neuritis with ataxic symptoms, are some of the complications which I have noted.

Several patients have become insane, at least half of them having melancholia, but most of them have been cured in time. They were all predisposed to insanity by family history, syphilis or alcoholic habits.

The neurasthenia which so often follows influenza is probably due to a granular degeneration or some other change in the cells of the highest nerve centres.

Pathology.—After Pfeiffer's discovery of the specific bacillus, Dr. Kaufmann found it at Kasr el Ainy in sputa and also in the bronchi of foundlings who had died of influenzic broncho-pneumonia at the homes of the wet nurses. The bacillus grows best at blood temperature, 37° C., and lives for 24—36 hours in ordinary water, but is quickly destroyed by dessication. It can be found for weeks, or even months, in sputum voided by a patient if the cough and expectoration persist, and it retains its infective power in moist sputum outside the body for 14 days. It has been seen in the nasal and bronchial secretions, the bronchial mucous membrane, the lung substance,

pleura, peribronchial and subpleural lymphatics, pericardium, endocardium, liver, spleen, kidney, middle ear and central nervous system. It has occasionally been found in the blood, which accounts for its diffusion to so many distant parts.

Diagnosis.—Where it is possible, a positive diagnosis can be made by the discovery of the specific bacillus in the sputum. This is almost the only way to differentiate true influenza from the severe *catarrhal cold*, sometimes called influenza or grippe, which runs through a household or a school. This spurious influenza is undoubtedly infectious, and probably dependent on an unknown microbe, but not on Pfeiffer's bacillus. During convalescence it will be noted that a patient regains his usual health in a few days after an attack of the spurious disease, but he may be many weeks recovering from the effects of true influenza. Under the heading of *enteric fever* I have referred to the difficulties of deciding between it and influenza. In Egypt there is one disease which requires special mention, because there are a very large number of points which are similar to both *dengue* and influenza. In fact, a few years ago, it was seriously contended by many in Europe who had never seen dengue that the two diseases were identical. In the following table I have contrasted them. In February 1897, an English lady, while convalescing under my care from dengue in Cairo, caught influenza, on the occasion of her first outing, from a patient suffering from that disease.

Prognosis.—There are practically no deaths among the Europeans in Egypt, because there are fewer, and less serious complications than in Europe, and the more favourable winter climate allows patients to be out of doors after a very few days confinement to the house. The daily presence of sunshine has also a very good effect on their spirits. The natives, however, die of broncho-pneumonia, especially in the case of uncared-for and insufficiently clothed young children. For instance, the general mortality in Alexandria rose in January 1890 from a steady weekly average of 172 to 245, the increase being due to deaths from influenza and its after effects.

H. H. the Khedive Tewfik Pasha lost his life from complications occurring during influenza. Dr. Engel says that the case mortality of those who consulted doctors was nearly one

	Influenza	Dengue
Distribution.	Spreads over whole countries with great rapidity.	Preference for sea-coasts, or for valleys of large rivers, and moves less rapidly.
	Not confined to mosquito zones. Met with in Iceland, Faroë Islands & Russia.	Believed to be introduced by mosquito puncture, never found further north than latitude 41° N.
Chief season in Egypt.	November to May. Likes damp cold.	August or September till December. Likes damp heat.
Incubation.	2 to 3 days.	4 days.
Symptoms.	Of protean variety. At onset, pain chiefly in head and eyeballs. Flushing of face and chest common.	No great variety. Pain in head and limbs at onset. Flushing of face and chest less common.
	Fever not always present, and, if present, often not high.	Fever always present, and generally high.
Eruption.	Rare.	Common, 1 to 6 days.
Desquamation.	Very rare.	Marked, lasting sometimes 3 to 4 weeks.
Pulse.	Rises with fever.	Remains low in spite of fever.
Vomiting & Gastric symptoms.	Generally absent.	Common.
Pain.	Neuralgic pains sometimes.	Pains in muscles, bones & joints are more severe, & may continue during or after convalescence.
Throat.	Larynx & trachea affected.	Pharyngitis & tonsillitis.
	Dyspnœa common.	No dyspnœa.
Convalescence.	Rapid after mild attack without complications.	Very slow. Patient prostrated, anæmic, thin and liable to pains for 2 or more weeks.
Complications.	Many & various, bronchitis, pneumonia, pleurisy, diarrhœa, syncope, herpes, otitis, diseases of nervous system etc.	Very rare.
Prognosis.	Doubtful in the aged, careless, and those with pre-existing respiratory disease.	Always favourable.

per cent, but his statistics must be drawn largely from hospital cases, for out of hundreds of private cases I have only lost one, a very old man with advanced kidney disease.

Treatment.—No greater lesson has been learned during the recent pandemic, than the folly of a patient trying to fight this disease by carrying on his usual occupations and exposing himself to all weathers. Prolonged illnesses have resulted from neglect at the onset of complications. The drug treatment, as in most other fevers, is purely symptomatic, and of the various tonics which are so necessary when the acute symptoms are over, I unhesitatingly put strychnia first. Alcohol, even in large quantities, may be necessary for patients suffering from heart or nerve symptoms, but the quantity must be prescribed accurately by the physician, and discontinued at the end of the illness. Change of air to the desert or to the sea does more for long standing cases than any food or drug.

Prevention.—Strict isolation where possible is the essential. Some require special protection from infection, such as the phthisical, the aged and the delicate. The sputum, handkerchiefs, and, later, the patient's room, should be carefully disinfected. During a pandemic, there must always be a special danger of contracting influenza in health resorts, where convalescents flock to cast it off. As the bacillus enters the body by the nose or mouth, it is prudent to irrigate these cavities, and further, the individual who wishes to try and escape infection, will do well to take quinine and a moderate amount of alcohol.

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PLAGUE.

Synonyms:—Oriental plague, Bubonic plague, Levantine plague, Pestis. *Fr.* Peste. *Ar.* el Taoun, Kobbah, Habouba.

HISTORY FROM EARLIEST TIMES TO 1853.

There is no mention in Egyptian papyri of this disease. At one time it was thought that one of the hieroglyphic words signified plague, but it has since been identified as tertian malaria. The ancients knew little of differential diagnosis and often called by the same name all fatal epidemic disorders. One of the earliest historical records of plague in Palestine, and of the connection between it and mice (or rats) is to be found in the Bible in I Samuel V—VI. It is not certain when these chapters were written, but probably about B.C. 600. Dr. Rufus of Ephesus quoted, about 100 A.D., from the writings of physicians who had correctly described plague in Egypt, Syria and Libya in the third century B.C. The first pandemic of the world's history was in the reign of Marcus Aurelius in the second century A. D., and the next was in the time of Justinian, when plague began in Egypt in 542 and spread to Syria and all over Europe where it lasted intermittently for 50 years. The pestilence is said to have begun at Pelusium, near the modern town of Port Said, and from thence reached Alexandria and Constantinople, where 5000 persons a-day are said to have died, and the Emperor himself suffered from it. When it attacked Europe it was found that it began at the sea coast and spread inland; this looks as if the disease was communicated by the passengers, rats or cargoes of sailing ships, and if it be true that the sea-ports of Egypt were first attacked, it is more than probable that they had been infected by some other country, and therefore do not deserve the obloquy of initiating this historical pandemic.

Many writers, including Pariset, the French Commissioner sent to Egypt in 1828 to study the plague, have surmised that Egypt was free of this disease until the 6th century, and that this date corresponds with the epoch when insufficient burial was substituted for the embalming of dead bodies. The objections to this ingenious theory to account for the constant presence of plague in Egypt, are very strong. (1) In archaic times, before embalming was introduced, there is no history of plague; (2) mummification began before the time of the Pyramid builders and lasted till about A.D. 400, its sole object being to prevent decomposition of the body, but in mummy times the poorest people must have been embalmed so badly that their bodies could not have been protected from decomposition; (3) long after mummification was given up, till as late as the 11th century, the Copts dried their dead by sun or by fire, for M. Maspero tells me he has found some artificially dried bodies of about that date at Ekhmin; (4) it is quite erroneous to suppose that plague during historical times was not present in Egypt before the 6th century A.D.

But whatever the causes may have been, there can be no doubt about the affinity which plague had with Lower Egypt for many centuries. Arab historians presumably refer only to those epidemics which were of startling malignancy. In 695, Abdul Aziz ibn Merwan, the Governor of Egypt, fled to Heluan to escape plague, and in 968—9, under the reign of Kafur, the eunuch, plague is said to have killed more than half a million people in and near Cairo.

In 1294 it is reported that 700 corpses from plague were carried out of one of the gates of Cairo in a single day, and again in or about 1412, the Sultan el Muayyed clothed himself in common white wool in mourning for the plague, which was ravaging the country.

But of all the great historical outbreaks, the third pandemic, usually called the Black Death, in the 14th century, has left its mark most deeply; it has been calculated that altogether some 25 million people lost their lives from it. It first became epidemic in southern Russia, spread slowly over the whole of Europe, and then remained epidemic there for at least two centuries. It reached Arabia, Egypt and Constantinople about

1346, and clung persistently to them long after it was banished from western Europe.

The epidemics all came from the east to the west, or from Mediterranean countries northward, and this led to the enforcement of quarantine regulations both by sea and land. Venice is said to have instituted the first lazzaretto against plague in 1403.

Hirsch gives the dates of 21 epidemics in Egypt between 1783 and 1844, when, according to all writers, plague suddenly and almost miraculously disappeared.

The French army in Egypt lost 1689 men from plague, 1300 of these deaths being between December 1798 and July 1799.

Desgenettes says that at first the sick were shunned, but later, when the disease became better known and was less fatal, there was no fear of contagion; he believes that there were 100,000 deaths from plague in Upper Egypt in 1800.

Captain Thurman, a French officer, tells us of plague spreading from Alexandria to Rosetta and Abukir in January 1799, while Napoleon was starting with 15,000 men for Syria. He not unreasonably complains that two months later, on leaving for Alexandria, he had to undergo 40 days quarantine in a miserable barrack, eating onions and biscuits. On arrival at Alexandria he found that the baggage he had left there was partly eaten by *rats*. On April 30 plague had ceased in his camp at Abukir, and he took his own measures of disinfection. The soldiers were unexpectedly paraded with all their accoutrements and were marched down to the beach, where every man was made to walk into the sea until he was breast deep.

Then, for two hours, everybody and everything, including the guns and the muskets, were washed in the sea. All rubbish, straw and old clothes were burnt, and the buildings were whitewashed, after which no more plague appeared, so that this method was strongly recommended to the sanitary commissions for use in the lazzarettos. Curiously enough, he says the plague never attacked the natives at Abukir.

On July 1st, 1799, he was again at Alexandria, and says that, although the army had lost 2400 men, including nearly all the "officiers de santé," there were no new cases there after St. John's day (June 24). Friends were no longer afraid to grasp each

other's hands, barricades were down, houses opened, and only some vessels in the Bay were still shy of visitors.

Ever since Prosper Alpinus, writers have told us of the interesting superstition that the miraculous *nukhta*, or dew, fell from heaven on St John's day, and that no one ever fell sick of plague after that date, and very few died. But the dew occurs all through the summer months, and when careful enquiry took place it was often found that plague continued well into July. The Turks, believing that the hour of a man's death is fore-ordained, did not trouble to shun society during epidemics, but the Christian inhabitants of Alexandria and Cairo shut themselves up in their houses while the plague was raging, disinfected their rooms regularly with sulphur, and purified themselves and all their belongings with water, if any member of the household became ill from any ailment, however trivial. But after St John's day was safely past, all prudence was thrown to the winds, ordinary visits were paid and business transacted, and, worst of all, the clothes of the thousands who had lately died of plague were carried to the market place to be handled, bought and worn by the public, without fear of danger. When one remembers that this clothing included furs and woollen garments, besides silk and cotton, one can guess that this misplaced confidence was one of the reasons which caused plague to cling so closely to Egypt and Turkey.

The inhabitants of the Levant at this time were specially afraid of infection from dogs, cats, rats and flies.

Sotira, who studied plague at Rosetta for three years, 1798—1800, was shocked at some of his colleagues not believing in the contagion of plague, but he did not himself believe in smaller animals suffering from it. He says that rats were very numerous in Egypt, and that they and the dogs used to eat plague corpses. Like other writers, he found that plague was specially fatal to negroes, and that the inhabitants of Alexandria, Rosetta and Damietta suffered much less than the French garrisons.

Desgenettes inoculated himself in Egypt unsuccessfully with pus from the bubo of a convalescent patient, with the object of raising the drooping spirits of the French troops. He says he had for more than three weeks two little painful inflamed spots in the groin and axilla where he pricked himself with the lancet.

In April 1801, plague broke out in Cairo among the recently arrived English troops. The unfortunate patients were confined in huts vacated by the French soldiers, and mostly died, while the doctors and attendants caught plague from them and also died.

Dr. White wished to discover if plague could not be checked or rendered less virulent by the introduction of inoculation and therefore inoculated himself at Rosetta in the autumn of 1801 with matter taken from plague buboes. The attempt entirely failed twice, but on the third occasion proved fatal, death occurring on the third day of the disease. Abercromby's force consisted of 18,000 men, of whom 380 contracted plague and 173 died between April 12th and August 26th 1801.

Clot says that in 1824, in Cairo alone, 30,000 people died of plague, though there were only two or three deaths in Alexandria.

Dr. Grassi, Chief Physician of the Sanitary Department in Egypt, wrote, in 1839, that Egypt was free of plague from 1825 to 1834, with the exception of an outbreak limited to Damietta between June and August 1832. On July 7th, 1834, a sick priest was seen by Grassi at the Greek convent in Alexandria, and it was then found that, 14 days before, plague had been introduced among the priests by a visitor from Cyprus. The linen of this visitor had already been sent to a negro hamlet to be washed and within six weeks 18 out of the 110 villagers had died of plague. The disease smouldered in Alexandria during the winter, but in March 1835 the death rate there rose to 200 daily. From Alexandria it spread all over Lower and Upper Egypt, and Lane tells us that it destroyed 80,000 people in Cairo, or one third of the population, and more than 200,000 in all Egypt, chiefly in the Delta, while Sir John Bowring calculated that 300,000 families were attacked.

At that time the medical advisers of the Egyptian Government, who belonged to various nationalities, but were mostly French or Italian, were divided in two hostile camps, those who believed and those who could not believe in the contagion of plague. Clot Bey, who was one of the leaders of the non-contagionist group, brought forward dozens of reasons for proving his view, some few of which may be quoted: He cites that in 1836, immediately after the epidemic, all the clothing and furniture of 50,000 people who had lately died of plague,

were sold in the bazaars without communicating infection to a single person; and that the clerks and workmen, who entered the 600 houses of the victims to make an inventory of the contents, escaped unscathed. Again, that in the Ezbekia hospital some 3000 plague patients were treated, and, after the epidemic, ordinary patients were admitted to this hospital and slept under the blankets, which (contrary to orders) had neither been disinfected nor aired since they had been saturated with discharges from plague patients, yet no new case occurred. Many women continued to suckle their children until they died of plague, without communicating it to their offspring, while, on the other hand, many infants at the breast died of plague without giving it to their mothers. He quotes the case of the wife of a European doctor, who suffered from plague at Alexandria without infecting her household whom she saw daily, the child she was suckling, or her husband, whose bed she shared during her illness.

At one period of this scientific campaign the contagionists taunted their adversaries with not having slept with plague patients, worn their clothes, lived with them, touched them, or inoculated themselves with pus from the buboes. Clot Bey says that he and his colleagues submitted themselves to all these tests. During the five months of plague in 1835 they visited numbers of patients in hospitals and in dirty huts, where they sat on the mats and rags of the patients, and allowed their own hands and clothes to be stained by "vomited matter, the blood of those bled, the pus of thousands of incised buboes, and the serum of carbuncles;" over 100 autopsies were made with patience and assiduity, and experiments were carried out on dogs, on condemned prisoners and on the doctors themselves. M. Bulard (a French chemist, who was, however, employed as a doctor) took the shirt off a dying plague patient and wore it for 48 hours, while Clot himself was twice inoculated with the pus from a bubo, and with the blood of a patient who died shortly afterwards. Of the whole band of workers only one (Rigaud) succumbed to plague. Again, in 1836, in the Cairo central hospital hundreds of plague patients were admitted, and although they were treated in separate wards when the disease was recognized, the servants and the linen were common to all

classes of patients, and yet the infection did not spread. The students of medicine of that day lived near Abuzabel and 11 of them out of 120 died.

The Viceroy had retired into quarantine at the Shubra palace, his household consisted of 300 people, of whom 8 were attacked by plague.

Clot Bey records the experiments made on four condemned prisoners: the first was dressed in the shirt and drawers still wet with the sweat of a plague patient, after which he was made to sleep all night in the patient's bed, four days afterwards he developed plague, of which he died in another four days. A similar experiment on a second prisoner resulted in his developing plague on the sixth day, when he got a bubo in the left groin, from which he rapidly recovered.

This unfortunate individual was inoculated twenty days later in the groin and armpit with serum taken from a plague carbuncle and, as this produced no effect, he was again inoculated a week later with plague blood without result. The third man was inoculated at the front of the right elbow with blood taken from the cephalic vein of a patient who had been ill for two days. He developed general benign symptoms two days later, and had a bubo which got rapidly well without suppurating. The fourth prisoner, aged 16, also escaped scot free, though he was inoculated with serum from a plague carbuncle, then with pus from a recent bubo in his armpit and groin, and finally, a fortnight later, this operation was repeated.

These experiments have some historic, but no scientific interest, for they only demonstrate that plague, especially the bubonic form, cannot always be communicated, and we have no knowledge of how the experiments were conducted, or if the bacillus in addition to pus was ever injected.

Deron was so convinced that plague was non-infectious, that he used to take his son, aged seven, with him to the hospitals.

Pruner's belief was that plague was spontaneously generated in Egypt without any doubt. "It develops in sporadic and epidemic form; in the first case it is not contagious, in the second it is probable that it becomes so in a similar way to typhus, malignant dysentery etc." He believed that plague was not a disease *sui generis*, but was only a fulminating typhus, and like

other writers of his time he points out that plague epidemics in Egypt were often followed by outbreaks of typhus in the spring. He found plague most vigorous from March to May, encouraged by an air temperature of 21° C., and destroyed by that of 38° C. In 1844 he recorded a few cases among the natives of Damietta and Alexandria, and in the Arnauts and other soldiers, but in 1845 he heard of none.

Kinglake visited Cairo at some time between 1838 and 1843, and described an outbreak there which was probably that of 1841. Most of the people with whom he had any transactions died of plague, including his banker, doctor, landlord, and donkey boy, within a period of less than three weeks. His banker (a Levantine), standing behind a grating, accepted his letter of credit with iron tongs! Kinglake did not hear of any plague patients in Cairo who recovered, and the mortality was said to be as high as 1200 a day.

Griesinger was in Egypt from 1850 to 1852, and saw no plague, he said that it had been entirely absent from the country for seven or eight years, in spite of many French writers, who maintained that the disease was still endemic in Egypt. He firmly believed in the infectious nature of the virus.

It has always seemed somewhat unaccountable that plague should have disappeared so suddenly in 1844 from its old home in Egypt, and I was interested, a few years ago, in finding a thesis, dated 1853, which tends to show that the subsidence of the disease was very gradual. The writer, Dr. Cuny, a Frenchman who had lately served the Egyptian Government as chief Sanitary inspector of Middle and Upper Egypt, says that after 1844 there were every year sporadic cases both in the Delta and on the Upper Nile. He cites one typical case out of many seen in Cairo in April 1853, but it appears that these sporadic cases were, as a rule, quite mild, occurring every spring with fever and swollen glands, which rarely suppurated, and though sometimes dangerous to life, were more often so benign that patients could go back to their work after a few days rest. In 1850 Cuny saw plague in several villages, and counted ten cases in one village alone, but one of the Government vaccinators was nearly dismissed from his post for giving him this information! The native doctor of Samalut excused himself

for not reporting plague in a rich townsman, by saying that he would certainly be assassinated if the dreaded land quarantine were imposed. Another native doctor, under Cuny, was dismissed by the Government because he had announced some cases of sporadic plague!

Dr. Rossi, at this time, besides a few other officials, was dismissed from the Viceroy's service because he had reported a sporadic case near Cairo. The people buried their dead secretly in their houses, mosques or churches, and feared even more than the plague itself the quarantine measures and the insatiable avarice of the sanitary officials. I have not been able to meet with any evidence of plague in Egypt between 1853 and 1898, but if Cuny is to be believed, plague disappeared quite gradually from Egypt, and not suddenly in 1844. There is an analogous belief with regard to the disappearance of plague from London, to take only one example. It is popularly believed that plague suddenly appeared in that city in 1665, and was banished by the great fire of the following year. But, as a matter of fact, plague existed in London every year (with four exceptions) from 1603 to 1679, so that the fire can have had very little to do with its cessation, though it doubtless destroyed many houses and barns infested by rats. (It is interesting to note that in 1665 the dogs, cats, mice and rats were all ordered to be killed in London.)

The disappearance of plague from Turkey and Egypt in the middle of the nineteenth century was, doubtless, partly due to the decline of trade in Baghdad and other towns on the Euphrates and Tigris, and to the appearance of steamers at Suez in and after 1839, which brought merchandise from India to Egypt instead of its coming from Persia and Syria. But this disappearance of the disease in an epidemic form followed immediately the introduction of sanitary measures into Constantinople in 1840 and into Egypt in 1842, and though these measures were far from being complete, they probably had some influence upon the general health of the country. One of the reforms was connected with burials, for, according to Griesinger, corpses, until this time, were not buried in Egypt, but were merely laid on the ground and covered with dust.

He states that plague used to break out in Egypt in the

neighbourhood of a cemetery if the earth was disturbed. It was about this time that burials were forbidden in private houses, and cemeteries were established outside the towns away from the prevailing wind.

HISTORY FROM 1854 TO 1894.

During these forty years, plague was confined to certain endemic centres in Asia and North Africa, for Europe had been free since 1841, when the disease was last known to be in Constantinople, and America and Australia had never been attacked.

(1) In China plague was present, certainly since 1860 and probably much longer, in the south-western province of Yünnan, bordering on Thibet and Burma. The plague in this centre became extremely active in 1893, and spread, for the first time in history, to Canton and Hongkong in 1894, and thus began the great modern pandemic. There were other outbreaks in China, but none so important as in the Yünnan area.

(2) In India plague has been endemic since 1823 in Kumaon and Garhwal on the southern slopes of the Himalayas, but the disease slumbered very mildly between 1892 and 1896, so that it is almost certain that the present epidemic in India is due to an extension of plague from China, and not from this centre in the Himalayas.

(3) In Mesopotamia plague was present in 1867 and later years in the Hindieh marshes of the lower Euphrates, near the centre of pilgrimage for Shiah Muhammedans, and in Baghdad it has been epidemic on several occasions. In 1892 it was also prevalent among the Arab tribes.

(4) In Persia it existed in several districts between 1863 and 1890.

(5) In Siberia plague seems to have been present for many years in the Transbaikal province, where there was an epizoötic disease which attacked a kind of marmot. This marmot plague is intensely infectious, for, according to Clemow, it attacks persons who touch the bodies of animals that have died from it, and from them it spreads to other human beings.

(6) In Eastern Mongolia, some twelve days ride from Peking,

plague has been epidemic at least since 1888, and here again attacked the same marmot.

(7) In Russian Central Asia, near the borders of Samarkand and Bokhara, a disease, which is, apparently, plague, has occurred since about 1878.

(8) In south-western Arabia there has been an endemic area in the Assyr Hills since 1816, but the outbreaks seem to be confined to a strip of country, mainly occupied by the Beni-Sheir and Beni-Amr tribes of Arabs. There is no evidence that the disease has been conveyed by Bedawin from these hills to other parts of Arabia, or still less, to Egypt.

(9) In Africa the most important endemic centre is in Uganda on the north-western shores of the Victoria Nyanza. The disease is locally called "kaumpali," has been described by many travellers and was bacteriologically identified with plague by Koch in 1898. As communication is now established between Uganda and the Sudan, I may here say that this district is occasionally the scene of a very high mortality, as, for example, in 1898—9, when 467 deaths from plague occurred out of a population of only 715 in Bukoba, adjoining the German territory. Another sharp epidemic occurred in Buddu, to the south of Uganda, in 1896, and since then it has been carried by native traders to various islands on the Victoria Nyanza. It will, however, be afterwards seen that plague has always been introduced into Egypt from its sea-ports.

(10) In Tripoli plague was epidemic in Benghazi in 1858 and 1874, and since then typhus has often overrun the province.

(11) Only once during these forty years has plague appeared in Europe. In Astrakhan and six villages on the Volga it was present between 1877—9, but it soon died out, and showed no tendency to spread.

Attention has only of late been directed to these various endemic areas. Considering their number it seems wonderful that no general epidemic occurred before 1894.

HISTORY SINCE 1894.

The fourth pandemic in the world's history began in 1894, and is still continuing. From the Yünnan endemic centre

plague attacked many other parts of China, Formosa and Japan. It reached the city of Bombay in 1896, and ever since that year there have been serious epidemics there between December and April. It has practically spread all over India, though some districts have shown themselves more resistant to it than others, and in spite of its sojourn for eight years, it shows no sign of declining. The deaths officially reported from India in 1904 were 1,040,429 from plague, while in April 1905, they reached the enormous figure of 56,732 in one week! Jedda became infected in May, 1897, and again in 1899, probably by traders from Bombay. In Mecca there were two cases recorded in 1899, and others in Yambo in 1900, but Medina has never yet been attacked.

In 1902 the disease broke out at Nairobi in British East Africa. It occurred in Madagascar and Mauritius in November 1898, being the first time that the southern hemisphere had ever been attacked, and this disposed of the theory previously held that plague could not exist south of a certain latitude. It has also been reported from South America, the Pacific Islands, Australia and San Francisco, where it occurred in 1900 among the Chinese.

In South Africa it was first seen among natives from India in the Transvaal in February 1899, it spread to several of the towns, but there was no great extension of the disease during the Boer war, as might have been expected.

In Europe plague was seen at Oporto, in July 1899, among the dock labourers who were probably infected indirectly from some of the Portuguese colonies, and in the same month it appeared in the pneumonic form at Kolobovka at the mouth of the Volga. In 1900 it was at Smyrna, Syria, Constantinople, Batum and Odessa, and in this year and the following one infected ships caused local outbreaks at Glasgow, Liverpool, Naples, Marseilles and other ports.

HISTORY AND DISTRIBUTION IN EGYPT SINCE 1898.

The old plague traditions in Egypt were sufficiently well known to create no little alarm when the disease was officially announced in Alexandria in May, 1899. It was impossible to

decide how Alexandria became infected, but it is most probable that the disease was imported from Bombay by infected rats or cargo. The outbreak was not severe in Alexandria, but it lingered throughout the summer and autumn, reappearing on several occasions after the disease was believed to have been stamped out.

The timorous members of the Levantine and Jew population were not overcome by panic as they usually are during a cholera epidemic, for they saw that plague chose its victims chiefly from the poorest natives and Europeans. They also comforted themselves with the loudly expressed assurance that this modern disease was something quite different to the classical epidemics of plague. It is hardly necessary now to state that there was no foundation for this belief, clinically the disease in Egypt resembles absolutely the plague raging in India, and the accounts of the plague in all former epidemics, and when the bacillus was discovered, bacteriological examination showed that there was only one disease, whether in India, Egypt or elsewhere. It was then pretended for a time that the bacillus in Egypt was of an attenuated strain, and therefore the urgent measures ordained by the Government were unnecessary, but the mortality in Egyptian cases is sufficient in itself to disprove this theory.

In 1897 the Egyptian Government had sent Sir John Rogers, Dr. Bitter and Ibrahim Pasha Hassan to Bombay in order to study the epidemic on the spot, with a view to arranging measures for the protection of Egypt. Their report pointed out that the general sanitary conditions which prevailed in Egypt were much worse than those of Bombay, so that if plague once became established a greater mortality might be expected. The measures they recommended were the immediate isolation of the sick, the placing under observation for five or six days of those in direct contact, and the efficient disinfection of the house, effects and clothing of the sick person. These measures were adopted when plague reached Egypt, and with certain improvements have since been continued. The stamping-out process has, on the whole, been very successful, and no serious complaints have been made on the part of any class of society, for in certain ways connected with religion and customs the conditions have been easier than in India.

In May, 1898, Dr. Creswell discovered a man suffering from plague at Suez; he was a fireman employed on a steamer which had been used for the transport of pilgrims, and he had just returned from Ras el Aswad, a port some seven miles to the west of Jedda. All the officers and crew of the steamer were fortunately still in Suez, and they were sent to Moses Wells to undergo a quarantine of six days. The passengers who had arrived by the steamer were traced and kept under observation in their homes for a period of ten days, and they all continued in perfect health. Towards the end of the quarantine period of the officers and crew, the doctor of the steamer fell ill with an axillary bubo and died of secondary pneumonia, while two other members of the crew also suffered from the disease, but recovered. The steamer was then thoroughly disinfected, and no further cases occurred either at Suez or in any other part of Egypt. This is an interesting story as showing the value of early and accurate diagnosis of the first case of human plague arriving in a non-infected but susceptible country. There is no evidence that the cases in Alexandria in the following spring could possibly be attributed to the infected steamer at Suez, a year before.

In 1899 the first batch of pilgrims, returning from the Holy Places, only arrived at Tor to undergo their fifteen days quarantine on May 3, so that the Alexandria cases cannot have been occasioned by pilgrims returning from Jedda and Mecca, which were then both infected.

On May 2, 1899, Dr. Valassopoulo discovered a young Greek suffering from plague in Alexandria, and similar cases were soon found in the Greek and Jewish hospitals. There is some evidence for believing that the first case of plague occurred in all probability in Alexandria during the early part of April. The first known twenty cases were confined almost exclusively to the Greek assistants of grocers and bakers, and it was not till later that the disease extended to the Egyptian population. In June, two Frenchmen were brought to the Government hospital suffering from plague, and one of them stated that he was employed in the rooms of the French mill, where the empty sacks were placed on their return to the mill from a shop in the infected quarter of the city. He always wore open list

slippers, and it is possible that his inguinal bubo arose from some abrasion of the foot coming in contact with an infected sack. The director of the mill, on being questioned, remembered that he had noticed a great mortality among the rats some fortnight earlier, but since that time these animals had entirely disappeared, so that it was impossible to obtain any for examination. In the Moharrem Bey barracks five policemen out of 160 contracted plague, though none of them had been on duty in the infected part of the city; here also, at least three rats had been seen, dead or dying, in the barracks a fortnight before the outbreak, and it is worth mentioning that the policemen were accustomed to walk about the barracks with bare feet.

Soon after the plague was declared in Alexandria, public notices invited the inhabitants to give information about dead rats, and a small fee was paid for those brought to the laboratory for examination. In this way several hundred rats and mice were examined, but in only two instances were the animals found to have died of plague; one was a mouse found dead in the house of a person who had died of plague, the other was a rat found near the Greek hospital, where several cases of plague had been treated. Curiously enough, none of the rats found in the grocery stores and bakehouses of the original Greek patients were found to be infected with plague.

It was during this summer that Dr. Gotschlich made his interesting discovery that, in pneumonic plague, the bacillus can remain in the sputa so long as the 76th day, or 48 days after the temperature has become normal, and be still sufficiently virulent after that lapse of time to kill a guinea pig; but on the 82nd day bacilli had entirely disappeared. In a second case, virulent bacilli killed a guinea pig on the 41st day, being 33 days after the fever was over, and in the third case the animal was killed on the 35th day, or 20 days after the temperature had subsided.

In all, from May 20th to November 2nd, 1899, there were 96 cases of whom 46 died; 68 of the cases were natives, 28 were Europeans. The number of persons isolated was 672, and the precautionary measures were continued in spite of the cessation of plague. Only one other town in Egypt, Damanhur, forty miles to the south of Alexandria, was infected in

1899. Two cases occurred, one was traceable to goods imported from a plague house in Alexandria, and the other occurred in a railway porter who had transferred goods from trucks to various stores. Many suspicious cases were notified from other towns, but bacteriological examination invariably showed that they were not plague.

In 1900, at the end of April, two Greeks and a Syrian died of plague at Port Said, and it was then found that several other Europeans had died there during the month of illnesses diagnosed by the European doctors as "influenza with glandular swellings," "cerebral influenza" or "pneumonia." There was no doubt that some of these patients had died of plague which had not been recognized by their medical attendants. It is suggested that plague on this occasion reached Port Said by means of infected clothing from Aden, Jedda or Yambo, all of which were then infected ports. The disease continued in Port Said until July, and accounted for 35 deaths there. In May a new outbreak began at Alexandria in the person of a female rag picker, who was found dead in one of the quarters largely occupied by warehouses. Dropping cases occurred until November, and caused 20 deaths. In view of the obstinacy with which the disease adhered to certain quarters it was decided to experiment with a system which had not been before adopted for financial reasons. This was the copious and systematic washing of the floors, and, to a certain extent, the walls of all houses, cellars, stores and stables with a solution of 1 per 1000 of corrosive sublimate. A disinfection service was organized for Port Said, and the whole town, European and native, was thoroughly disinfected in this way. As the work progressed, no single case occurred in any part of the town which had been subjected to this treatment.

The rubbish of all the dirty houses was carted away in sacks to be burned with petroleum, and an interesting proof occurred of the apparent value of this measure. One of the men in charge of the destructor, in spite of warnings, insisted on working with naked feet and legs, and in a few days he was admitted to the plague hospital with typical inguinal buboes, apparently contracted directly from the rubbish.

During May 1900 three cases of plague were reported, two

proving fatal, at Suakin, and rats were observed to be dying in some numbers, but there was no further spread of infection.

In 1901 the total number of plague cases throughout Egypt was 206, and of these 104, or 50·7 per cent died. The cases were distributed over 8 different towns, Alexandria, Port Said, Zagazig, Minia, Mit-Gamr, Benha, Zifta and Tanta. This was the third year in succession that Alexandria had been invaded, and it became evident that the disease had established itself there. The first case occurred in a native at Gabbari, who had worked in market gardens, and who, possibly, had become infected by plague rags found in the garden manure. The cases lasted from April till November. In Port Said the patients were admitted between June and September, and during August a considerable number of dead rats were found in the native quarters of the town. At Zagazig plague was present from June to August, and an excessive mortality among rats was observed, besides interesting proof of their migration during the outbreak from their accustomed haunts to neighbouring gardens. In Mansura there was only one case which occurred in the person of a man who had travelled from Zagazig.

Only five cases occurred at Minia in June, but the first appearance of plague in a town in Upper Egypt created some apprehension, as it was naturally feared that this might be a sign of general infection of the whole country. Minia is about 300 miles from Alexandria, and 150 miles south of Cairo. At Mit-Gamr there were 20 cases (11 deaths) between August and November, and the first case was that of a Greek who had been employed at a bakery, where rice and flour from Alexandria were being used. Dead rats were found in the infected houses, and bacterioscopical examination showed that they had died of plague. At Benha there were only five cases with three deaths during September, the infection apparently having been transmitted from Mit-Gamr. During the disinfection of the house in which the first of the recognized cases occurred, several dead rats were found, which had died from plague. At Zifta there were only ten cases, but eight of them were fatal between October and December. The infection reached Tanta in December, and caused an epidemic which continued till April, 1902.

a man, accompanied by his wife and three sisters, went across the Nile to attend a relative's funeral, three days later the man, his wife, two of these sisters and two other sisters had all died from plague. Later on, other cases occurred in the same village, but all were confined to the family or neighbours of the original case.

Great difficulty was experienced in Upper Egypt by the Sanitary Inspectors and their staff, for bare-faced concealment of patients was the rule rather than the exception, and cases were eventually found in houses which had been carefully searched a day or two before. For instance, plague was suspected at the village of Naknak, but on inspection no cases could be found; yet, on the following day, the wife of the sheikh of the village died of pneumonic plague and on the next day his niece and two other people were found plague stricken in the village, there can be no doubt that these cases were purposely hidden during the house-to-house inspection. Although this hamlet contained only 12 houses, there were 24 cases, 17 pneumonic and 7 bubonic. A disinfector and a woman nursing the sick both died of pneumonic plague. The Egyptian peasants far away from centres of civilization do not in the least believe in the necessity of cleanliness and disinfection, they imagine that all illnesses come from an evil spirit, and that the Government officials are their hereditary enemies. Typhus was present during the outbreak at Girga and Baliana, in the latter town there were 33 cases of this fever with 22 deaths. At Naga el Atiat, near Deshna, there were in May 35 cases of plague, all pneumonic, and all fatal. In Minia and Beni Suef provinces the plague, on the contrary, was almost entirely bubonic, the total number of cases was 275, of which 122 died, giving a mortality of 44·3 per cent. Only three of these cases were pneumonic, though three others developed secondary pneumonia. This local outbreak was remarkable for the number of children attacked. Out of the 275 cases, 95 were below the age of ten, and twelve of them occurred under the age of one year. The age of the youngest patient was one month, (right cervical bubo) that of the oldest was a woman who was said to be 95 years old.

In these outbreaks about one-third of the cases were relatives

or next door neighbours of previous cases, and another interesting peculiarity was the multiplicity of the buboes.

In July, Dr. Unsworth died of bubonic plague at Zagazig, being the first of the Sanitary Inspectors who had contracted the disease since plague first reached Egypt in 1899. He died on the fourth day of the disease of heart syncope, shortly after jumping out of bed at a sudden alarm of fire in the next room.

Plague has been entirely absent from the Sudan and there is no record in past history of its ever having advanced further south than Wady Halfa. This is sufficient in itself to disprove the suggested theory that modern plague in Egypt has spread from the Uganda centre.

There have been no cases in the English Army of Occupation, or among winter visitors, and practically none among the richer classes of society.

CAUSES.

Bacteriology.—The plague bacillus was discovered by Kitasato and Yersin in 1894 at Hong Kong.

It may be demonstrated from post mortem specimens or from punctures of suspected glands, by making cover glass preparations, drying, fixing with a mixture of equal parts of ether and absolute alcohol, and staining for a few minutes with methylene blue. After washing, drying and mounting, the preparation can be examined.

As was first pointed out by Bitter, no plague bacilli are visible after suppuration has taken place. The bacillus is killed by heating for ten minutes at 62°—65° C., and loses its virulence by drying. It is killed instantaneously by corrosive sublimate 1/1000, in ten minutes by carbolic acid and lysol 1/100 and in five minutes by sulphuric acid 1/1000. It is killed in direct sunlight in four hours, in ordinary drinking water in three days, and in sterilised water in eight days.

Presence of the bacillus in man during life.—It is found in buboes in large quantities, and to a less extent in the œdematous fluid surrounding the inflamed gland, which is nearly always hæmorrhagic; the smear, after staining, shows pus cor-

puscles (in advanced cases), cell débris, blood corpuscles and micro-organisms.

In about half the number of living cases, bacilli can be found in the blood, provided a thick film be used and the bacteriologist is an expert, but if the blood be not very carefully taken from the ear or finger it is very apt to show bipolar staining bacilli, which are not plague bacilli, but probably B. coli from the surface of the skin. In pneumonic cases, smears from the sputum will reveal the bacillus almost in pure culture.

Post mortem smears from most of the internal organs, especially the liver and spleen, show the organism; it is even more likely to be found in the blood of the heart, above the aortic valves, and it is sometimes found in the retroperitoneal glands.

Accounts are on record of successful direct inoculation experiments from man to man, in addition to Clot Bey's condemned prisoners. The outbreak in the Vienna laboratory is conclusive, because there was no other plague in the country. In October, 1898, the attendant who had charge of animals inoculated with experimental plague, died of the pneumonic variety, and bacilli were found in his sputa. The doctor who attended him also died of pneumonic plague, and two nurses contracted the disease, but recovered. Again, in 1903, an Austrian doctor, who was studying bacteriology in Berlin, accidentally wounded his hand while injecting a rat with a culture of plague bacillus. He contracted plague himself and died, and the hospital attendant who nursed him also became ill. It has long been shown by Dr. Bitter that the plague bacillus produces a hæmorrhagic septicæmia, and that, in animals of great susceptibility, it passes directly into the blood without any visible local reaction taking place. Plague, however, is different to other septicæmic diseases in that the local reaction, which occurs in man and other animals not of the greatest susceptibility, sets in, not at the point of inoculation, but in the corresponding lymphatic glands.

According to Dr. Bitter, we should consider plague in the first place "as a local infection, chiefly of the lymphatic glands, which often ends in a general septicæmic infection. The bacillus enters some small abrasion of the skin but usually shows no local signs at the point of inoculation and infects the nearest

lymphatic glands in which it begins to multiply itself and cause by the resulting toxin the local and general symptoms of the disease. In a great number of cases it remains limited in the infected glands and does not enter the blood. These cases usually recover. The bacillus is killed in the glands by the anti-bacteria of the organism and the glands begin to suppurate. When the tissue of the gland is entirely or chiefly transformed into pus, living bacilli can, as a rule, no longer be found. In some few cases the gland does not suppurate. If the anti-bacteria are not strong enough to limit the bacillus to the glands, it gradually invades the neighbouring tissue and the blood, causing a septicæmia which generally ends fatally in a few days. As this septicæmia may cause hæmorrhage in all the mucous membranes and as the blood contains plague bacilli all the excreta of septicæmic patients may be infectious and ought to be considered as such. Embolic pneumonia caused by the bacilli is often present as a symptom of this septicæmia and the sputa in such cases contain numerous plague bacilli which are very dangerous as a means of spreading the disease. The particles of sputa from such a patient when inhaled by the healthy person can produce primary plague pneumonia without the lymphatic glands being affected."

But Dr. Hunter now suggests that plague is primarily a septicæmia, and if so, the fact of the disease subsequently assuming the bubonic or pneumonic form becomes a secondary matter. He maintains that bacilli can be found in the blood in bubonic cases before the formation of the bubo, i.e. during the first or second day of the illness.

Age.—Infants usually escape it, but many cases have been recorded between the ages of one and five years. A woman died of plague two days after giving birth to a healthy child, who did not contract the disease. Of 61 children under the age of 18 months, who were nursed by plague stricken mothers, only ten became infected. (Jennings).

The following figures are taken from statistics collected by the Sanitary Department. Of 556 cases from all Egypt, of which careful records could be obtained during 1904, 148 were under the age of 10, 135 between 11 and 20, 99 between 21 and 30, 77 between 31 and 40, 51 between 41 and 50, 29

between 51 and 60, and 17 were over 60. This shows very clearly the great prevalence of the disease in the young.

Sex.—Both sexes are equally affected. When pregnant women are taken ill, they usually miscarry and die.

Nationality.—The Sudanese and Berberines are specially susceptible to plague, and have a high death rate. Europeans enjoy a greater immunity than the Egyptians, but this is, probably, dependent on social, not racial differences.

Occupation.—Doctors, nurses, attendants and others engaged on plague work, rarely contract the disease, but in addition to Dr. Unsworth, a few native disinfectors who were too careless to wear boots have succumbed. Post mortem examinations are attended by some risk.

The incidence of plague is not dependent on any trade, but men working in grain and grocery stores, infested by rats, are specially liable to be attacked.

Climate and season.—Climate has very little to do with the causation, and some other factor besides heat must be responsible for the absence of plague from the Sudan. It is, however, mainly a disease of temperate and sub-tropical countries, preferring a temperature which is neither very hot nor very cold. In India, where the plague losses are calculated at nearly four millions of individuals since 1896, neither the temperature nor the humidity of the air have any influence on the prevalence of epidemics.

It is quite possible that the cessation of an epidemic, such as occurs in Egypt during July, has no direct relationship with the advent of warm, dry weather, but is chiefly dependent on the breeding season of rats, or the prevalence of fleas.

I have shown in the historical sketch that plague may now exist in Egypt during any month of the year, and none of the inhabitants of to-day believe that it will completely disappear in June. But there has been nothing of late years to upset the old belief that the disease, during epidemics, reaches its maximum in March and April, subsides after July and again becomes virulent in December or January. During the years 1834—43, there were 12,282 cases reported in Alexandria. Of these 403 per thousand occurred in March, 239 in April, and only one per thousand in September and October. The Nile

has nothing to do with the appearance or disappearance of the disease, except that a low Nile would conduce to want and filth, both of which are predisposing factors. It must not be forgotten that the cold and wet months drive people to sleep indoors in overcrowded, unventilated huts, which may have something to do, as in typhus and relapsing fevers, with the maximum prevalence in the spring months.

In any given locality, there may be a few cases of plague all the year round, but it is only at a certain season that the disease becomes epidemic. This season varies in different localities, but it is nearly always constant in the same locality. For instance, in Northern Europe, the usual season is late summer and autumn, in Asia Minor it is in spring and summer, in countries south of the Equator plague becomes epidemic at a different time of the year to Europe, but always in the corresponding season. Cape Town and Sydney have their epidemic season, like Egypt, in March, April and May. It is evident that the epidemic season is governed by some periodical law at present unknown to us, and if plague recurs in any place in an epidemic form, it is almost invariably at the same season of the year as was the preceding epidemic. (Simpson).

Again, the duration of an epidemic also runs a certain course in spite of the presence or absence of preventive measures. Simpson has pointed out that there is, first of all, a period of hesitation more or less prolonged, then a sudden but fluctuating rise, which reaches its height in the course of about three months, then comes a fluctuating fall, which is much more rapid than the rise, and, finally, the disease lingers on for some months in a sporadic form.

Immunity.—One attack conveys only doubtful immunity, and there are cases reported from other countries of individuals suffering twice within a year from plague.

MEANS OF SPREAD OF THE DISEASE.

(1) *Connection between plague in rat and man.*—Sambon has pointed out that this connection was well known to the ancient Greeks who, in Asia Minor, worshipped Apollo the Rat, as "the god whose arrows spread the plague," and was, at the

same time, "the destroyer of rats." This divinity was represented on monuments treading on a rat. In Roman times, when Æsculapius replaced the more ancient god of medicine, he was represented with a rat at his feet and a small naked figure standing by with arms outstretched; this can be seen in the British Museum on a coin of Lucius Verus, struck at Pergamum.

Avicenna (Ibn Sina) wrote in the 11th century, that when plague was approaching "mice and other animals which live underground fly from their holes and stagger from them like intoxicated animals." But he says nothing about death among rats, and he does not hint that rats can in any way convey the disease to man.

Later writers have often quoted Ibn Sina, but added nothing on the subject from their own experience, until Savonarola wrote in the 15th century that mice and other animals which habitually lived in holes were found dead during plague epidemics. It must always be remembered that the ancients gave the same name to mice and rats. Natives of India have known for hundreds of years that it was unsafe for people to remain in a village if the rats sickened and died, but, on the whole, very little attention was paid to the question of plague affecting rats as well as men, until the present pandemic. Koch reports that the natives of Uganda leave their homes if they find dead rats in a plague locality. In former epidemics it is possible that rats were not so numerous, or were not affected, or that the disease among them escaped the attention of historians. Today it seems to be established as a fact that they are the cause of *some* epidemics, that human plague breaks out about a fortnight after rats have been noticed to be dying, and that the disease among human beings subsides some two weeks after all the rats have died or migrated. Rats, too, are the probable cause of the mysterious cropping up of human cases at unexpected times and in unsuspected places, and are often responsible for keeping alive the smouldering embers of endemic, chronic plague, which are capable of bursting into an acute epidemic form when circumstances become favourable. The rat theory also easily accounts for the obstinate clinging of plague to a particular area, and its periodical recrudescence in that area during the breeding season of rats.

It has been found that people living on the ground floor of a house are more often attacked by plague than their neighbours who live on the upper storeys of the same house, and in mediæval epidemics it used to be said, "plague does not go upstairs."

The Indian Plague Commission summarized their views on this point as follows: "It has been clearly established that when the infection of plague has been imported into a town or village, rats have, in many cases, contracted the infection before any indigenous case occurred among men. Again, it has been clearly established that, where rats have become infected, they have been instrumental in spreading plague in villages, and in carrying the infection outward from evacuated quarters. Hardly less clear is it, that rats have been frequently at work in disseminating the infection of plague in inhabited towns and villages, and that in some places they have been responsible for the occurrence of an explosive outbreak of plague among men. It is, however, important to note that the outbreak of plague among rats has not in all cases been followed by the occurrence of plague among men, and that explosive outbreaks have sometimes been due to causes other than the spread of infection by rats."

It cannot too strongly be insisted upon that the rat, when he is infected, usually suffers from the septicæmic form of plague, that his excreta are widely distributed throughout dwelling and store houses, and that the bacillus occurs in the urine, fæces, pus from buboes and bronchial secretion of plague rats.

Infected rats probably travel only short distances on foot, but they may spread the disease to neighbouring villages or to the unaffected parts of an infected town. They may also travel concealed in bales of cargo, and may thus proceed long distances by train or boat. Even if a rat dies on the journey a man handling the contaminated bale which contains the carcass, has been known to have caught plague.

The disease is believed to have been imported into South Africa by infected rats in South American forage, and the dock labourers in various ports were the first to be attacked. Again. in Sydney, plague is believed to have been brought by foreign rats, who first infected the rats at the dock and subsequently

the town and its people. Many other ports have been infected by incoming rats. The infection in these ports has always first attacked the dock labourers or grocers, which goes some way to show that it is not the crew or passengers of a ship who are the usual carriers of the disease. But there are many instances, on the other hand, of plague having been conveyed in this pandemic to new countries by human beings. To give only one instance: A ship reached England in 1901 from Alexandria, which brought with it nine cases of plague, two of which had become infected while attending upon the sick.

Enough has been said under this heading to show the importance of medical inspection of shipping from infected ports, provided always that not only human beings, but also ship rats are examined before they have time to escape.

Many instances have occurred in several parts of the world of plague being conveyed to unaffected countries by means of ships. The disease has generally begun in the person of a fireman, stoker, or some other member of the crew likely to have come in contact with the rats on board, and dead or sick rats have often been found on the vessel.

Plague ought now to be studied more particularly by those interested in epidemiology and natural history, for one of the first questions we want settled is whether all species of rats are equally susceptible, and if not, which are the most dangerous kinds and which are the various types to be met with in any given country. It is obvious that plague is most likely to spread among rats at the time of the greatest frequency of their young, for the young rats are even more susceptible to plague than the full grown ones. Dr. Gotschlich has examined 6500 dead rats to see when their breeding season occurs. He found in Alexandria that the maximum number of rats with young was in May and June, and that only one per cent of them were pregnant between October and February. It will be important to know how many breeding seasons during the year the rats in Egypt have, but we already have the interesting fact that the time for greatest production corresponds with the maximum of epidemic prevalence of plague in man.

The rat infected with plague shows the symptoms of acute hæmorrhagic septicæmia. The inoculated animal becomes dull

and crouches in a corner of its cage, emitting, from time to time, short squealing sounds, there is complete loss of appetite, ruffling of hair, mucous discharge from the mouth, diarrhœa and fever. On handling it, there is general tenderness of the body, especially in the abdomen, the reflexes are exaggerated, and sometimes there is twitching of the muscles of the limbs. Shortly before death, which occurs between the third and fifth day, the animal appears unable to move, irregular convulsions take place, during which the urine and fæces are voided, coma gradually supervenes, and death occurs during a convulsion. The post mortem results are: congestion and extravasation in the subcutaneous tissues, œdematous enlargement of the superficial lymphatic glands, and sometimes the presence of small buboes. When the animal has been inoculated through the skin, there are, at the point of inoculation, swelling, œdema and infiltration of the connective tissue with blood and bacilli. The blood itself is fluid and dark, the muscle of the heart is in a condition of granular degeneration, the lungs are full of dark-coloured thick blood, and the liver, spleen and kidneys are all congested. The mesenteric glands are enlarged and inflamed, and there are, sometimes, hæmorrhages between the layers of the mesentery and behind the peritoneum. Bacilli are found in the spleen and most of the organs of the body, and in the blood, urine, fæces and saliva.

Chronic rat plague.—This is sometimes met with during the intervals between epidemics, and can only be detected by persistent examination of thousands of rats during the whole year. (Gotschlich).

During life the rats are usually thin, their hair is ruffled, they eat little and often have a discharge of mucus from the mouth and slight diarrhœa. Death takes place from exhaustion and at the autopsy the animals are extremely emaciated, but the organs and tissues do not show the marked congestion of the acute type. The glands are often enlarged and hard, bacilli are found chiefly in the spleen, which is always enlarged, and, occasionally, in other organs. Hunter says that in Hongkong about 75 per cent of the rats caught alive and found infected suffered from chronic plague.

Plague can be artificially induced in rats by subcutaneous

inoculation, by rubbing the virus into the skin of the shaved abdomen, by intra-peritoneal inoculation, by inhalation (producing primary plague pneumonia), by smearing plague material on the mucous membrane of the conjunctiva and the nose, and by feeding. The most important mode of spontaneous infection of the disease by the rat is by its mouth, and may be direct from rats by contact, food, urine, fæces or saliva, or direct from man by means of his excreta, or from food, or from contact with infected houses, ships, clothing and cargo, or from other vermin, such as mice, ants, flies and fleas.

Rats often eat the dead bodies of their comrades, and also mice.

The rats examined in Sydney belonged to two species, the brown (mus decumanus) and the black (mus rattus). There were about as many of one as of the other, but the infected specimens were nearly all among brown rats. The brown rat is a great traveller, practically omnivorous, and wonderfully prolific. In Europe several litters are produced annually, each of which generally contains from eight to ten, and sometimes as many as twelve young, and a female will breed when only half grown, though the number of its progeny is then but three or four at a birth.

2. *Connection between rat-fleas and man.*—Even among those who now believe that human plague is often a primary disease among rats, there are many who search for the reason of the inefficiency of plague rats alone to cause plague in man. It has been found that handling live rats has been followed by plague, but that it is safe for children to play with those which have been dead for some time.

The general freedom from infection of attendants in plague hospitals and of disinfectors makes it difficult to believe that fleas can be the chief intermediary for human infection. However, in Sydney, Tidswell noticed that during the rat plague, fleas were abundant on all the rats (healthy and sick), which were brought to the laboratory, whereas fleas were very rare on rats brought at the close of the epizoötic. The rat bugs (cimex lectuaris) continued to occur in the same proportion during and after the epizoötic. Here again we are in need of information about what kind of rat-fleas we have in Egypt, how many of them will bite man, and will they bite all through the

year? The flea of man (*pulex irritans*) has, in Cairo, a very well defined biting season, from January or February to May or June, whereas in India the insect is troublesome all the year round, though always worse during the rains.

Tidswell examined 300 specimens of rat-fleas from different parts of Australasia, and found they consisted of 4 species, *pulex pallidus* 169, *pulex fasciatus* 66, *typhlopsylla musculi* 58 and *pulex serraticeps* 7. Of these the three varieties of pulex will all bite man, and the last named has been found on various animals including rats, men, cats and dogs. Similar proof that some fleas will bite both rat and man, has been made by Raybaud and Gauthier at Marseilles. Probably most fleas, when starving, will bite man. Though it is not yet proved that the flea can convey the bacillus from rat to rat, or from rat to man, so that this insect can hardly be the carrier of epidemic plague, it is obvious that an agent, endowed with locomotive powers, able to conceal itself under protective clothing and capable of penetrating the epithelium without causing either distinct pain or a visible wound, is highly worthy of our attention. Plague bacilli were found in fleas by Ogata in 1896, and since then by observers in other countries.

3. *Infection from man to man.*—Pneumonic cases, both primary and secondary (septicæmic), are certainly the best instances of this form of infection, and sometimes tend to reproduce themselves during an epidemic. They are infectious from the beginning, because the sputum contains enormous numbers of bacilli. Bubonic cases are, at first, but little infectious, but upon the development of lung symptoms or septicæmia, they may become highly infectious. Plague is always a very infectious disease in houses which are insanitary, badly ventilated, or infected by rats.

4. *Clothing.*—Clothes or bedding can retain infection for a long period. The bacillus can survive in flannel, cotton and linen which have been allowed to dry naturally.

In India there has been a heavy incidence of plague among the washermen, and in some cases plague has been brought to a country by contaminated clothing. In some outbreaks, second hand clothesmen, or rag pickers, have been the first victims.

5. *Soil.*—Though the bacillus cannot usually be recovered

from the soil, there can be no doubt that it is present in plague houses on the surface of mud floors, from gross contamination by sputum or excreta of rat or man, and, at one time, the Sanitary Inspectors added to their previous regulations the necessity of removing the upper surface of the mud floors of plague-infected huts. This measure is too costly for universal application, but is now reserved for houses where several cases have occurred.

6. *Water.*—There is no evidence that plague has ever been a water-borne disease.

7. *Insanitary Houses.*—There can be no doubt of the fact that plague clings closely to certain infected houses, and from historical times both rats and men have known the importance of escaping from infected localities. The ill ventilation and the general absence of hygiene in the dwellings of the poorer Egyptians are among the predisposing causes. Again, too, it must not be forgotten that insanitary conditions are often associated with the prevalence of rats.

MODES OF ENTRY.

Plague can enter the human body by (1) inoculation, (2) inhalation and (3) by the tonsils.

1. *Inoculation.*—Probably the most common method of entry of the plague bacillus is by the skin, through abrasions such as wounds or scratches or through the bites of insects. It is not absolutely necessary to suppose that the flea itself introduces the bacillus, for it is obvious that the bacillus might enter through the bite, or the patient might infect himself by scratching the bite with a contaminated finger.

It is more difficult to explain the fact that in Europeans, who usually go about booted, it is the femoral and inguinal glands which are chiefly affected, as in the case of the bare-footed natives, but it must be remembered that every European is barefooted when going to bed at night or when getting up in the morning, and if his dwelling house is overrun with septicæmic rats his foot might easily come in contact with some of their excreta. As a matter of fact, many of the Europeans stricken with bubonic plague in Alexandria or Port Said during

the last few years, have been found, on examination, either to wear no boots at all while at their work, or such bad ones as to offer little protection.

The Indian Plague Commission found that there was a distinct relation between the area of skin surface draining into a particular set of glands, and the frequency with which those glands were affected. They showed that the areas of skin surfaces which drain respectively into the neck, arm-pit and groin have the ratio of 1 : 1 : 8·5, while, by hospital statistics in India, it was proved that the numbers of primary buboes in the same regions was 1 : 1·3 : 5·8. It would therefore appear that the more frequent occurrence of buboes in the groin is due to the fact that a much larger skin surface drains into these glands. To continue the investigation, one would now like to know whether rat-fleas are more apt to bite the lower extremities of man than other parts of his body. In this connection it is apparently true that the human flea is more apt to bite his victims in the lower extremities than in the rest of the body, provided that the victim is walking about.

There are instances of inoculation of plague by cutting the fingers at post mortem examinations, or through abrasions already existing, and in such cases a bubo has developed in the neighbourhood of the site of inoculation.

Simpson (p. 256) says that there is a case on record of a pneumonic patient spitting on to the conjunctiva of a nurse, who developed conjunctivitis, a bubo below the ear on the affected side and died. As a rule, it is quite impossible to discover the point of inoculation, and this is equally difficult in some other diseases, such as tetanus or erysipelas of the face.

2. *Inhalation.*—This is the mode of entry in all cases of pneumonic, and some cases of septicæmic plague. Dark, dirty and densely crowded houses, with an ill-ventilated atmosphere are specially dangerous, for it has been found that the air for about two metres in front of a pneumonic patient is teeming with bacilli. The loss of infectivity of plague in the open air or in circumstances of perfect ventilation, is another point closely resembling the poison of typhus. Experimentally, plague has been produced in rats by placing a small quantity of plague culture on their nasal mucous membranes.

3. *Tonsils.*—The comparative frequency of cervical buboes in children is thought to be due to their habit of putting things into the mouth, or by direct infection through the tonsil. It must be remembered also that dirty children have many abrasions in their scalps due to the irritation of lice and consequent scratching.

Some workers at Hongkong have insisted upon the existence of a primary intestinal infection, but this is not usually recognized. Experimentally, infection has been contracted by animals fed on plague-infected food. (Simpson.)

Symptoms and Course.—The three chief varieties of plague are (1) bubonic or glandular, which may remain a simple local infection, or may become septicæmic, (a symptom of which is pneumonia), (2) septicæmic from the beginning of the disease, and (3) pneumonic.

(1) Bubonic type. The incubation is generally from two to four days, but may vary from one to seven days. Some cases of it are so mild that the diagnosis can only be made bacteriologically by puncturing the gland, these are the so called cases of *pestis minor* or *pestis ambulans.*

When premonitory symptoms are present, they are slight, notably anorexia, headache, insomnia, malaise, tenderness and pain in the inguinal or axillary region; these symptoms may last for a few hours or two or three days.

But the invasion is usually sudden, and is ushered in by rigors, prostration, nausea, vomiting or diarrhœa, and, in some cases, the appearance of the bubo may precede the rigor. In little children one of the first symptoms is a convulsion. On the second day the temperature has already risen to its height, which may be 39°, 40° or even higher, but this usually falls to normal between the fifth and seventh days. The bubo, which is a term used for a collection of glands, is generally in the femoral region, but may be in the inguinal, and is 4 to 5 c.m. below Poupart's ligament. The bubo in the groin occurs in 75 to 80 per cent of all bubonic cases. Next in frequency come the axillary buboes, with about 10 per cent incidence, and, more rare, are the supra clavicular. The parotid buboes vary very much in different epidemics, and are sometimes about three per cent. The popliteal, retroperitoneal, cervical, sub-

maxillary, occipital and elbow glands are among those rarely affected. The submaxillary glands in children, however, are more commonly selected because of their habit of putting their fingers and everything else into their mouths. The bubo swells rapidly, so that in one or two days it has reached the size of a hen's egg, and may become as large as a man's fist, while the patient is obliged to lie with his limb flexed. The bubo sometimes ends in resolution, but, more often, in suppuration between the eighth and tenth day; severe cases die on or before the seventh day. Usually only one group of glands can be felt, but there may be a double glandular infection.

In Egypt in 1904 out of 480 bubonic cases, 449 had single, while 31 had multiple buboes. As regards cervical buboes, children under the age of five gave a percentage of 48·6, between 6 and 10 years this dropped to 19·4, and after 10 years to 11 per cent. By an inverse ratio, the percentage of buboes in the groin rose from 19·7 in children under five years, to 36 between 6 and 10, and 57·7 after the age of 10.

The site of the bubo did not differ much in the two sexes with the exception of the inguinal and femoral regions, where men suffered to a greater extent.

The general symptoms are as follows: the face flushed and anxious, the skin hot and dry, eyes sunken and early emaciation, the conjunctivæ injected, the speech thick and hesitating, with confusion of memory, thirst, vomiting, diarrhœa or constipation, and headache which is chiefly frontal or temporal. There is evidence of dilatation of the heart, and sometimes of heart failure, the pulse is small, weak and often irregular, beating 100 to 140 times in the minute, the respirations are hurried, varying from 30 to 50. The tongue is at first moist and coated, but soon becomes dry and fissured, and sordes appears on the lips and teeth. As Professor Bitter has pointed out, the tongue shows two characteristic longitudinal white strips, while the centre and edges remain red. Most patients suffer from giddiness and staggering gait, and delirium, when present, begins on the second day, and is worse at night. The urine is scanty, and contains a trace of albumen and granular casts. The spleen is always increased in size, the liver is sometimes enlarged, jaundice is rare. Very rare, also, are petechiæ and hæmorrhages

in an ordinary case, but some give a history of spitting blood. If there is any secondary fever after the seventh day, this is generally due to suppuration of glands, or to pneumonia. The convalescence is very slow, and may take from five to eight weeks if the bubo does not suppurate. Though abrasions are sometimes present between the toes, it is usually impossible to find any trace by which the bacillus has entered the body, but it may be assumed that the bacillus infects the lymphatic glands nearest its point of entry. In many cases the poison appears to remain in these glands, and does not enter the blood, in which case the prognosis is good. If the bacillus, however, is not limited to the lymphatic glands, it gradually invades the neighbouring tissues, enters the blood current and produces secondary septicæmia, which generally kills the patient in a very few days.

(2) Septicæmic form. The percentage of this variety varies in different epidemics, and may be differently classed by individual observers. It is characterized by early delirium and coma, uncontrollable vomiting, hæmatemesis, hæmaturia, hæmorrhages in the skin, convulsions and the presence of bacilli in the blood. Women, in addition, suffer from uterine hæmorrhage. The patients may die in a few hours or from one to two days after the onset. These are the classical cases of the Black Death and other epidemics, the patient being apparently well one morning, finding evidence of hæmorrhage in the afternoon, and dying before next morning. In Alexandria, of late years, there have been some instances of sudden death in the street of people who were not known to be ill.

The urine and fæces in this form are infectious and ought to be treated carefully. I have already said that rats get the septicæmic form, which accounts for their high mortality; their urine and fæces often contain bacilli, and soil the ground, which is walked upon or slept upon by natives of the poorer class.

(3) Pneumonic form. Some plague epidemics in India have been called "cough illness," and there have been many records of this type in historical times, but it was not recognized as a special variety till 1896 in Bombay. (Childe). The onset is sudden, like an ordinary croupous pneumonia, but this is found to be lobular, not lobar. In some epidemics there are no cases, in others a

perceptible amount, the percentage of all cases varying from one to five. In exceptional outbreaks they may form the chief variety and tend to reproduce themselves. The sputa are coughed up easily, and are generally profuse, consisting of serous muco-pus, which may, however, be rusty brown in colour. The respirations are quick, but there is not the same disproportion of ratio between the pulse and the respiration as is seen in ordinary acute pneumonia. The temperature varies between 39° and 40°·5. The lung shows indefinite physical signs, there may be dulness over one or more lobes, or no dulness, while moist sounds are generally heard at both bases. The lymphatic glands are tender, but not increased in size perceptibly; the spleen is slightly enlarged, the patient suffers from gradually increasing prostration and delirium, and usually dies on the fourth day from failure of the heart, sometimes accompanied by coma.

The sputa contain very numerous plague bacilli, almost a pure culture, and are very dangerous as a means of spreading the disease, in fact, this is the chief way in which plague is transmitted directly from man to man. When the bacilli are locked up in a gland they cannot do much harm to other individuals, but if they are floating about in the air, which others breathe, a whole household may be infected by the expectoration without a single lymphatic gland being attacked. During one winter at Tanta there were 11 cases in one house, and the whole percentage of the primary pneumonic cases was as high as 30 of the total number. Another danger about lung cases is that when an individual does recover he may still have living bacilli in his sputa for a period of three weeks. Many of us have seen healthy looking convalescents in the Greek hospital at Alexandria indignantly protesting at being still kept in the wards, though every day their sputa showed countless bacilli, which were powerful enough to kill guinea pigs, and yet the stethoscope revealed almost no abnormal sounds. Such a person could easily transmit plague from one town or country to another in spite of all quarantine regulations. Some of these lung patients show hæmorrhagic sputum for only one day, and it then becomes white and innocent-looking mucus, like the expectoration of ordinary bronchitis, yet it teems with plague bacilli.

The different varieties of plague are sometimes confusing to those unacquainted with the disease, and I have, therefore, been accustomed, in my lectures, to cite the somewhat analogous case of the tubercle bacillus. In tuberculosis the virus may be locked up in the cervical or other glands, which used to be called scrofulous or strumous, or it may chiefly affect the lungs, or it may be disseminated throughout the whole body by means of the blood stream in the form which we call general tuberculosis.

Complications.—The most common are: uræmia, nephritis, paralysis of the vagus nerve, aphonia, aphasia, blindness and deafness. Then there are the secondary pyæmic conditions evidenced by abscesses, boils, carbuncles, gangrenous erysipelas, parotitis and parenchymatous keratitis. The commonest sequelæ are thus either nervous or suppurative.

Mortality.—Bitter maintains that the true mortality of hospital cases in Egypt is between 35 and 40 per cent, and these figures correspond with the statistics in some of the hospitals in India and elsewhere. Gotschlich points out the difference in mortality between the summer cases from April to October, when the disease is chiefly caused by rats and produces a human death rate of 45 per cent (including cases found dead), and the winter cases between November and March, when the much higher mortality of 72 is reached. Of 54 patients treated in the Greek hospital in Alexandria in 1901—2, only 11 died which gives a mortality of 20.3 per cent. On the other hand, in India, there is often a death rate of 60 to 90 per cent, because many cases are not under Government control, and those mild ones which recover are not included in the statistics.

Death may occur at any stage of the disease, but most frequently from the third to the fifth day, from heart failure, hæmorrhage or suffocation, when the cervical region or the tonsils are affected. Sex has, apparently, no influence on the mortality, but there is certainly a higher death rate with coloured than among white people.

Morbid Anatomy.—There is well marked post mortem discolouration on all the dependent parts of the body, though, of course, it is impossible to determine the cause of death by this inspection alone. Emaciation is not a characteristic sign, because

the majority of cases occur in well nourished individuals. The temperature is sometimes subnormal at death, but in cases which die feverish the temperature is often raised after death, and may be as high as 42° or 43° C. in the rectum. When petechiæ are present on the skin they are most frequently met with about the groin and anus, the axillæ, or the mucous membranes of the mouth and nose. The skin may also show papules, vesicles or pustules, which, when large, may be called boils. True carbuncles are not found as the direct result of plague.

The bubo is found to be a series of congested glands embedded in a mass of extravasated blood, with extensive exudation into the adjoining areolar tissue. The individual glands will vary in size from that of a pea to a walnut, and, when cut into, they are of dark purple colour. In the groin the swelling is most likely to be femoral, but it may also be inguinal with extension to the iliac, lumbar and hypogastric regions, and the hæmorrhagic infiltration may extend down the thigh to the popliteal space, or upwards into the scrotum, or into the pelvis. Next in frequency come the axillary buboes, from which the œdema and hæmorrhage may extend into the surrounding tissues of the scapular, pectoral, intercostal and cervical regions. The cervical buboes occur most frequently in the submaxillary, submental, supra- and infra-clavicular, retro-auricular and parotid regions. All the lymphatic glands of the body, if carefully examined, will be found to be somewhat affected, even in cases of pneumonic plague.

On cutting open a plague corpse, one's eye is at once caught by the congestion of the subcutaneous tissues, in which may be found small or large extravasations of blood. The pericardial sac is of a reddish colour and contains a small quantity of blood stained serum. The heart's condition will vary with the number of days the patient has been ill, sometimes the whole organ is flabby, in others the left ventricle is firmly contracted with a dilated condition of the right ventricle. The blood is dark, thick and liquid, but some clots are found on the right side of the heart. The endocardium is normal in uncomplicated cases, except for the presence of small petechiæ on both sides. The myocardium is always in a condition of

degeneration, and petechiæ are sometimes found between the muscular fibres. The blood contains streptococci and an excess of leucocytes. The tonsils often show great swelling, with changes similar to those found in an ordinary bubo, and it is quite possible in these cases that the virus has entered the blood stream through this channel.

The pleuræ often contain a small quantity of blood-stained serum, they are usually congested and show petechiæ in varying numbers. The lungs are œdematous and hyperæmic, especially in the lower lobes, whilst hæmorrhagic infarcts are sometimes found. Hyperæmia and œdema are also present in the œsophagus, stomach and small intestine, and the peritoneal surface of the intestines is reddish. The liver is increased in size and weight, and extravasations of blood may be found scattered within it. The gall bladder is usually distended with thick, tenacious bile, from which bacilli have been isolated in pure culture. The spleen is usually from two to four times its normal size, the kidneys are hyperæmic, and the brain and its membranes, like the rest of the body, show general congestion and hyperæmia.

In septicæmic cases, the glands and the periglandular tissue are specially œdematous and hæmorrhagic, and there will be signs of hæmorrhage in some of the following parts, the stomach, intestines, kidneys, urinary passages, serous membranes or skin.

In pneumonic cases, the spleen may be of normal size, but broncho-pneumonia or pleurisy will be present, and there will be hæmorrhagic patches, surrounded by œdema, in the lower lobes of both lungs. The appearance is that of a typical lobular pneumonia, and a sticky, stringy juice exudes from the cut surface of the hæmorrhagic patches, which are blackish red in colour. Under the microscope the alveoli in the pneumonic areas are found to be entirely filled with blood and bacilli.

DIAGNOSIS.

Orientals and Europeans will all conceal the early cases of an outbreak, and little help can be expected by the Sanitary officer from any of the inhabitants, however intelligent. The Egyptians are specially clever in hiding their sick in the

fields, or their dead in their houses, in boxes under their beds, and even in disused ovens. The official must learn to suspect any village where there have been sudden deaths or an increased mortality, or any house where more than one case of pneumonia has occurred within a month, or any locality where rats are sick or dying.

Confusion has occurred in Egypt and elsewhere when the early cases of an outbreak have not been diagnosed by the doctors, who were unacquainted with the clinical symptoms of plague. The diagnosis of a suspected case can always be made by a bacteriologist, who will find bacilli in plague glands, if suppuration is not too far advanced, or in the sputum of a pneumonic case. The bacilli are always present during the first week in the fluid aspirated from a bubo, and are generally absent during the second week, but have been found as late as the 18th day. A rapid method of detecting plague bacilli in the blood is to take a blood film by Ross's method, making the film a little larger and thicker than for malarial examination, when, after decolorising and staining, the bacilli can be found. If the microscopical examination does not settle the diagnosis, cultures should be made on agar or in bouillon and then experiments must be carried out on guinea pigs. If the bacteriologist is unable to give a decided opinion after staining smears obtained from the patient, he must wait for three or four days till his animal experiments are completed. During this interval of time all necessary measures must be taken, as if the suspected case were one of declared plague, and diligent search must be made in the neighbourhood for other cases; the latest death returns should be overhauled, and all information should be obtained about the suspected case and the possibility of dead rats in the patient's house.

But it will seldom happen that the clinical observer who meets with a suspected case can at once obtain the co-operation of a skilled bacteriologist. The following is, therefore, the regular procedure in Egypt: after isolating the patient, the hands of the doctor are to be sterilized, and he should obey these instructions.

"The syringe together with the needle should be sterilized in boiling water, immediately before being used.

The syringe should be placed in a recipient containing cold water in such a quantity as to allow the syringe to be entirely covered with water. The water will then be heated to boiling point and kept boiling for 15 minutes. Should the piston of the syringe not fit well, the screw at the extremity of the piston rod should be well tightened. It is necessary to ascertain that the syringe works properly before it is sterilized.

Before the puncture is made the skin to be punctured should be first cleaned by rubbing it thoroughly with soap and water and a piece of clean cotton wool. Then it should be rubbed with cotton wool wet with corrosive sublimate (1 in 1000) and finally with cotton wool wet with pure alcohol.

The liquid withdrawn into the syringe must be deposited on the surface of the agar by pressing the piston. The operation should be carried out with great care in order to avoid contaminating the agar by other bacteria.

The cotton wool plug of the tube of agar should never be touched with the hand except on the end which is outside the tube.

The cotton wool plug should under no circumstances be placed on the table or on any other place.

The tube before being put into the wooden box should be well wrapped round with cotton wool in order to avoid its breaking during transport. These boxes should always be posted as ordinary letters and not as parcels.

Immediately after use, the syringe should be sterilized again in the following manner:—

Fill and refill the syringe several times with water withdrawn from the sterilizing vessel; this must be continued until no trace of blood or pus remains in the syringe.

During the emptying of the syringe the needle opening must always be kept beneath the water, to avoid any small drops of infected matter being by chance spread about.

The syringe and needle are then placed in the sterilizing vessel and the water heated to boiling point and kept boiling for 15 minutes.

The syringe is to be used only for puncturing suspicious cases of plague."

The Sanitary Department telegraphs the result of the examination, for which no fee is charged.

The differential diagnosis will seldom be difficult during the height of an epidemic, but at the beginning and end of one the clinician must be dependent on his bacteriological colleague. A *venereal or simple bubo* is generally inguinal, and not femoral, and there will usually be something in the history to aid one. *Chronic tubercular glands* have been mistaken for plague buboes, but here again the history should help. If, in addition to a bubo in the groin, a tender mass of glands is felt above Poupart's ligament, the case is likely to be plague. *Croupous pneumonia* is lobar, instead of the lobular plague pneumonia, herpes on the lips is never present in plague, the prostration in plague is out of all proportion to the physical signs, and the typical rusty, tenacious sputum of ordinary pneumonia is absent in plague, while, of course, the sputum should be examined for bacilli without delay. *Influenza* may be quite difficult to diagnose because the character of the pneumonia and of the nervous symptoms are very similar, but bacteriological examination of the sputum will at once decide the point.

Typhus is the disease, in my opinion, which is most like plague, but the fever in it lasts for two weeks, and the bubo, when present, is generally parotid, and does not appear during the first week, while, in patients with light coloured skins, there will be a typical eruption towards the end of the first week. Clot Bey, who was well acquainted with plague in Egypt, once visited a fever hospital in London, where he was shown several cases of typhus with parotid buboes, and he remarked "in Egypt we should call those cases plague."

Puerperal fever can be diagnosed from septicæmic plague by examining the blood, and by remembering that the plague symptoms precede delivery or abortion.

Egypt has, independently, accepted the dictum of the Indian Plague Commission that "no practical value attaches to the method of serum diagnosis."

PROGNOSIS.

No wise man likes to prophesy during the first week that any given case will get well, but if the patient survives until the sixth day there is a good chance for him. The pneumonic

and septicæmic cases are almost all fatal, though I have already mentioned three convalescents from the former in Alexandria. Hæmorrhage of all kinds, including petechial eruptions, are ominous. Pregnant women always miscarry, and generally die. The condition of the heart and kidneys before the attack is very important, and the presence of tubercle in the lungs is a serious danger. On the whole it is found that children and old people succumb more readily than strong adults. Much depends on the social condition of the patient and the nursing available, but, in India, many doctors and nurses have died of pneumonic plague in spite of devoted nursing in good hospitals. Cases attacked late in an epidemic should be milder than those at the beginning or at the height of the outbreak, but there are a good many exceptions to this rule. The lowered death rate during an outbreak is partly due to more cases being discovered, and to greater control being exercised over "contacts."

TREATMENT.

This is chiefly symptomatic, never forgetting the importance of preventing the spread of the disease to others. The patient should be put at once to bed, and must continue in the recumbent position to prevent syncope, until his heart shows no signs of weakness. Stimulants must be freely administered by the mouth, alcohol, ether, camphor, ammonia, or digitalis, whilst strychnia should be given hypodermically. In hospitals, if it were possible to obtain them, oxygen inhalations should be very valuable. The pain of buboes can be relieved by an ice bag or by belladonna and glycerine, suppurating glands must be incised with every precaution to prevent the spread of bacilli. The bacillus can be eliminated from the body by means of the sputum, urine, and fæces, all of which require special treatment; the suppurating gland must be considered infectious so long as it is discharging, though probably it no longer contains bacilli. Hyoscine, in doses of one hundredth of a grain will benefit sleeplessness, and vomiting may require hydrocyanic acid and the sucking of ice, while very obstinate vomiting necessitates rectal feeding. The diet must be liquid, while the temperature is raised, and later must be fairly light.

Yersin's serum was found useful at Glasgow in 1901, and in some other epidemics. In some mild cases subcutaneous injection alone will suffice, but in severe cases the combined method must be used early, and large doses employed from 60 to 200 c.c. The combined method means subcutaneous injection into the lymphatic area, which drains towards the bubo, and, at the same time, intravenous inoculation into a vein such as the median basilic. The temperature in several cases, after this treatment, fell from 40° or even 42° to normal, and remained below 37°, while the buboes ceased to be painful and did not suppurate. This serum is prepared by injecting living cultures of plague bacilli intra-venously into a horse, and later, bleeding the horse; it is prepared by the Pasteur Institute in Paris.

PREVENTION.

Patients suffering from plague or suspected plague are to be isolated, and the family and friends kept under observation, for the key of all anti-plague operations is isolation and practical and thorough disinfection. It is found very effective to enforce isolation of "contacts" of all *undeclared* cases of plague for this distinctly encourages the announcement of cases. When plague has been declared in a village, the inspectors are usually content to take the names of the "contacts" and visit them daily for five to ten days. The following summary shows some of the instructions issued by the Egyptian Government Sanitary Department, whose success in controlling the spread of plague is universally recognized.

The means necessary for efficient disinfection are: (1) a properly trained band of men and (2) a properly arranged equipment of tools and materials. Fifteen men under a trained disinfector are found to work well, all being provided with boots to wear. The equipment comprises a hoe, rake, brooms, sacks for carrying away rags, a box for dead rats, sublimate solution (concentrated 1 in 5) in kegs, a measure, carbolic acid, a pump, watering pots, buckets, disinfecting sacks, and two carts, where these are available. From the kegs a solution of sublimate (1 in 1000) is made and kept in half barrels or tubs and two of these are then attached to each gang. One of the men

is a rat hole filler, provided with a trowel, a pail containing mortar, a basket of broken glass and an iron rod one metre long, pointed at one end and bulbous at the other. With the sharp point he prods and finds the runs, after which he rams the broken glass into the hole with the blunt end of the rod and seals it up with mortar. One cart carries the equipment of the gang, and conveys the sacks of rags to the burning station, while the second carries the swept refuse to the same place. The burning station is selected either from a recognized rubbish place, or from some low-lying land, which wants filling up, or an old lime kiln may be utilized. Those who burn the refuse, like all other workers, are not only given boots, but are made to disinfect their hands and feet with a sublimate solution. The following is the method adopted on the discovery of a case of plague: A sick person still alive is removed to the hospital, unless the Sanitary Inspector gives permission for the case to be treated in a private house. A dead body is washed by the special attendant for this purpose (of the same sex as the corpse, to satisfy Muhammedan requirements), in a solution of sublimate (1/1000) and enveloped in a shroud saturated in a similar solution. The people who have been in contact with the sick person and the inhabitants of the house are given a bath of sublimate (1/2000) and clean clothing, while their own is disinfected. Where a hospital exists, it is convenient to send the contacts there for disinfection, but if no hospital exists, this must be carried out in a house in separate rooms for men and women, under proper supervision. The gang of men is then brought to work on the house concerned in the following way, bearing in mind that disinfection is rendered more easy and more perfect if the house is comparatively empty. All portable furniture is removed to the roof and courtyard, or to the street, while all bedding, pillows, carpets, curtains and cushions are put into the sacks for sending to the disinfecting machine. In small villages, where no such machine is available, these articles must be either burnt or soaked in sublimate solution, or exposed to strong sunshine for five days. Compensation must be made for all articles destroyed. Personal clothing, including shirts, drawers, sheets, mats etc must be wrung out in sublimate solution and then put on the roof to dry. All the rags, old

matting and burnable rubbish must be placed into the (kohna) sacks for conveying to the burning station. All dead rats and mice should be collected, placed in the solution of carbolic acid in the tin provided for the purpose, and burnt at the end of the day. The house must then be swept out from above downwards, and the sweepings carefully collected and loaded on to one of the carts. Following immediately upon the sweeping members of the gang come the disinfectors, who are distinguished by their special bands of coloured calico tied round the arm; they begin to disinfect the floor and the walls to the height of one metre with sublimate solution (1/1000), but when a pneumonic or septicæmic case is suspected the whole of the walls, up to the ceiling, are disinfected. The water pots are used for drenching the floor and walls, which, at the same time, are scrubbed with stable brooms. In better-class houses the pump is used for spraying the walls and floors. When these steps have been carried out, the "special" disinfection is completed, and the house is left to dry. Care must be taken not to leave the sublimate in pools on the floors, but no case of human poisoning has as yet occurred from this cause in Egypt, though some fowls have succumbed to the poison.

It is often necessary, in addition to this thorough disinfection of the house in which a case of plague has occurred, to proceed to a prophylactic disinfection of large areas in the neighbourhood of infected houses, or even throughout a whole town, but it is seldom necessary or practicable to carry out the whole series of steps mentioned above in such a case as this. The measures of such "general" disinfection as are applicable on a large scale, are: the emptying and cleaning of houses, especially as regards the dark, damp, unventilated cellars or rooms inhabited by the poorer classes, thorough disinfection with sublimate of all floors and staircases, and the maintenance of the household furniture and effects in full sunshine on the roof for a period of five days. It is advisable, in addition, to supplement the disinfection by whitewashing in cases of pneumonic plague and sometimes as an extra precaution in very dirty quarters where only bubonic plague has been met. The whitewashing gang consists of 17 men under a leader, provided with poles and barrels for mixing, buckets, brushes, a trowel, a quantity of lime

freshly burnt and a small quantity of *homra* for making mortar. The following points should be noted as regards whitewashing: the lime must be "quick", and only slaked at the time of use, one part of quick lime to five of water is the correct proportion in ordinary cases, and where time is of some value, it is sufficient to whitewash the walls to the height of 1½ metres. The part whitewashed should be thoroughly done, no matter how uneven the surface of the wall, no single part of it should be seen through the lime, and the floor, at the angle between it and the wall, should be also limed for a distance of 20 c.m. from the wall. All rat holes should be filled up with mortar and stones. In the case of *esheshes* (collections of dirty huts), especially if any pneumonic or septicæmic case has occurred in them, the whole of the inside should be limed, the roof removed, and thus left exposed to the sun for ten days at least. Finally, it must be especially understood that the measures are without value unless *thoroughly* carried out, for instance, care must be taken that the sublimate solution is of proper strength (1/1000), easily obtained by adding one litre of the 1/5 solution to the barrel of 200 litres of water. Similar care should be taken that the bedding, clothing and effects are really collected from the infected house; the native inclination to conceal goods is so common and so strong, that every means must be taken to defeat their action. Again, care must be taken, by means of a system of checks and receipts, that the rubbish sent out to be burnt is really handed over to the man whose duty it is to see it burnt. Also, it is not sufficient to place the bedding, clothing etc in a heap on the floor, each article must separately be spread out in the full sun and constantly turned. Finally, Sir H. Pinching reminds his subordinates that careful attention to these instructions will not only simplify and systematize the work, but will ensure an efficiency that could not be attained in any other way.

These various anti-plague measures would not be possible in Egypt if it were not for a Khedivial Decree dated May 27, 1899, which, when plague or cholera has invaded the country, gives the Sanitary authorities power to inspect any house where they believe that patients suffering from one of these diseases lie hidden. The same Decree gives them the power to transport to a temporary hospital the sick, and to watch over the contacts.

Needless post mortem examinations are discouraged by the Sanitary Department, partly to avoid the risk of scattering plague material, and partly because they are very unpopular with the natives.

L. Rogers, in Calcutta, found that neither the mineral acids nor permanganate of potash were reliable disinfectants of mud floors against the plague bacillus, even when used in solutions which are many times as strong as are required to destroy the bacillus in test tubes in 15 minutes; this was chiefly owing to the disinfectants being neutralized by the alkalis and organic matter contained in the earth. Phenol gave good results in the strength of 1 in 50, and so, also, did the acid perchloride of mercury 1/500.

During the presence of an epidemic in Egypt all assembling of crowds, whether for mooleds (fairs) or for pilgrimages, is discouraged by the Government.

Every effort must be made to destroy rats, whether sick or healthy, for even when they are not ill, they are a mischievous and useless pest to man. Rat-catchers should be employed wherever it is possible, and paid for their work, but care must be taken to prevent their introducing rats into their district in order to swell their returns. Spring traps made of wire are, apparently, the best, and should hold several rats; they should be handled as little as possible, and plunged into boiling water immediately after the rats have been removed from them, in order to keep them free from odour, as otherwise rats will soon learn to avoid them. The baits must be frequently changed, and may consist of toasted cheese, oatmeal, sun-flower seeds, herrings, or bread on which a few drops of aniseed oil have been sprinkled. The best poisons for rats are arsenic or phosphorus paste. A company has now been formed in England for making the Danysz rat virus, which is distributed on baits of bread; after eating it the rats become ill and die in five to twelve days. No animal has yet been found which can kill rats in large numbers and yet be itself insusceptible to plague.

Simpson rightly points out that plague measures should not cease so soon as the epidemic declines, on the contrary, they should continue during the period of quiescence in order to prevent a recrudescence.

Haffkine's Prophylactic.—A considerable trial of this remedy has been made in India, where the natives, especially in the Punjaub, willingly submitted to inoculation. But this method has been suspended by the Government of India, in consequence of an unfortunate mistake in a laboratory in 1902, as the result of which several deaths occurred from tetanus. The serum had evidently been substituted in error, and it was not unnatural that the natives should lose confidence in the measure.

Immunity after this inoculation, though never quite absolute, is, in the majority of cases, established in about ten days, and persists for a duration which is not yet determined, but for at least six months, and probably longer. Most of the medical officers and others who, of late years, have been on plague duty in India, believe in its prophylactic power, though it is obvious that each individual must undergo inoculation every year. The conclusions of the Indian Plague Commission upon this subject were:

(1) Inoculation is harmless.

(2) When given in the incubative stage, *i. e.* before the signs of plague are apparent, it has in many cases the power of aborting the disease. (This conclusion is by no means universally accepted).

(3) Inoculation affords to all those inoculated a strong protection against attack by plague.

(4) In the few cases where inoculated people are attacked, a very large proportion recover.

The remedy is prepared by cultivating plague bacilli in broth and clarified butter or cocoanut oil. After a lengthy process of five or six weeks, the bacilli are killed by exposing them to the temperature of 70° C. for one hour. The fluid is sealed up in vessels with a small quantity of some antiseptic, and then sent out for use. The usual dose is about 5 c.c. for an adult and it should be injected into the outer part of the upper arm.

A few hours after inoculation, reaction is evidenced by malaise, fever, and aching at the site of injection, followed by some prostration and redness and swelling of the upper arm. These symptoms usually last about 48 hours, when the patient may resume his normal occupations, though the local inflam-

mation may persist for some days and require fomentations of lead and opium.

It is said that by undergoing a second inoculation, ten days after the first injection, a greater amount of protection is secured. Haffkine's prophylactic has been favourably reported upon by officials in Australia and South Africa.

Since 1896 more than five million doses have been supplied to districts in India, besides more than half a million to the countries outside India. Among inoculated European residents no fatal cases of plague have, so far, been reported, and it is claimed that among the natives of India the number of attacks among the inoculated is less than one third of that in the non-inoculated population. The prophylactic material is now manufactured, not only in Bombay, but in London, Liverpool, Paris, Berlin, St. Petersburg, Berne and Pianosa in Italy.

QUARANTINE.

Land cordons are useless for all diseases and especially foolish in plague, where the short-sighted authorities put restrictions on human beings, without interfering with the rats who are at least as important as carriers of infection. Land quarantine never saved any country yet from any encroachment, and most of the great nations of Europe have now relinquished it. Railway trains and frontiers should, however, be inspected to prevent sick people travelling about, but no clinical examination can be sufficiently thorough to guarantee that a traveller has no bacilli in his body. The chief objection of English scientists to quarantine regulations is, that the faithful observance of these tends to prevent time, care and money being spent upon improving the hygiene of insanitary countries.

An International Sanitary Conference met in Paris in October, 1903, to consider how far anti-plague measures should be modified in consequence of our recently acquired knowledge of the disease, and the following paragraphs give a brief summary of the decisions of the Conference:

Five days (instead of ten days) after the isolation, death or cure of the last case of plague, a port is now considered as being free from plague. A harbour in which plague-rats have

been found is not to be considered as infected, as long as the disease has not attacked human beings. The Paris Conference, taking into consideration the facts collected by the Indian Plague Commission, decided that the incubation period of plague did not exceed five days as a rule, all the measures, therefore, were calculated on the basis of an incubation period of five days. The Conference recognized the fact that no merchandise was dangerous in itself, and, therefore it decided that no cargoes should be subjected to any measures, unless shown to be infected by the virus of plague or cholera. Under the heads of merchandise, however, the Conference did not include soiled linen, soiled bedding, and rags. Again, if merchandise has been in contact with plague rats, and if it cannot be disinfected, such merchandise may be kept in quarantine for ten days. Ships are classed as infected if a case of plague has occurred on board less than seven days before arrival. The presence of plague-rats on board does not imply that such a ship is infected, nor does it entail the application of the measures contained in this paragraph. Persons coming from infected ships may be quarantined for a period of five to ten days, or may be allowed to proceed to their own homes, but remain under medical supervision at home for a period not exceeding ten days; the latter is the British and the former is the Continental system. The rats on infected ships are to be destroyed. Ships are classed as "suspects" if there has been a case of plague more than seven days before arrival. The passengers of such ships may proceed to their own homes, but remain under medical supervision for five or ten days, and the ship is to be disinfected. Should the medical authorities think it necessary, rats may be destroyed on board the suspected ships, but on clean ships coming from infected ports the destruction of rats is optional. The destruction of rats may also be ordered on ships on which plague has appeared among rats only, and the same measure may be applied if an increased mortality has been noticed among the rats on board without there being an absolute proof that such rats died of plague.

The Conference also strongly recommended that in all large harbours permanent medical supervision of the sanitary state of the crews and population of the harbour should be enforced,

but it is doubtful whether, in Egypt, this measure can be carried out without increasing the powers now given to the sanitary authorities. It will be necessary for the Egyptian Government and the Quarantine Council to take measures for regulating the trade in old clothes at Port Said and Suez, and to control the health of the coal-heavers and crews there.

When the pilgrimage is clean, ships presenting certain favourable hygienic conditions and carrying foreign pilgrims may pass through the Canal in quarantine, after a medical visit, without undergoing quarantine at Tor; provided the Power to which the ship belongs has ratified all existing Conventions. Should the pilgrimage be foul, it is agreed that, when the Turkish quarantine stations have been improved, the Turkish pilgrims will no longer go to Tor but to Camaran.

The present arrangements for protection from returning pilgrims are as follows. On their return journey from the Holy places all pilgrims of all nationalities are stopped at Tor to undergo quarantine and disinfection according to regulations which depend upon the then state of health at Mecca. After leaving Tor the non-Egyptian pilgrims pass through the Suez Canal and are not allowed to land in Egypt. The Egyptian pilgrims, on the other hand, are visited by the male or female quarantine officers when the ship reaches Suez and if any cholera or plague be discovered on board the steamer all the passengers are sent back to Tor. If the ship be passed as healthy, she moors alongside the pilgrim wharf specially set apart for this purpose and divided off by barriers from the rest of the quay, and the pilgrims are allowed to land one by one, their passports are examined and their addresses are taken, and the list is forwarded to the sanitary inspector of the district to which they are going. As a second line of defence every pilgrim is then individually seen by Dr. Creswell, the medical officer of the port, who allows the obviously healthy to proceed without delay but keeps all the doubtful under observation for a thorough examination. After a detailed examination each man is either passed as healthy or is transferred to the isolation hospital where his disease is thoroughly investigated.

The Paris Conference did not consider it was justifiable to compel the destruction of rats in *all* vessels from plague-infected ports,

chiefly on the grounds that the risk of introduction to and spread of plague in European ports is small. But this risk is decidedly greater in Egyptian ports, unless great precautions are taken, and, instead of waiting to find out whether plague has occurred in any human being or in any rat upon a particular ship, it would be much better for sanitary reasons to assume that rats, on a vessel coming from a harbour infected with plague, are already "suspect", and should, therefore, be killed. No ship can be considered as disinfected if there are still any rats living on board. But the great practical difficulty is to know how to kill the rats efficiently without spoiling the cargo in the hold, or putting the ship owner to too great an expense. The Quarantine Board in Alexandria began to destroy rats on vessels in the harbour in July, 1901, and in the first month killed 1460 rats on twelve small craft. The cost was very satisfactory, only amounting to £4 to £6, per vessel for the sulphur, charcoal, quicklime, commercial carbolic acid and labour employed.

Haldane and Wade's report shows that the Clayton process for disinfecting ships and destroying rats has certain advantages and disadvantages. The apparatus consists of an iron furnace specially constructed to burn sulphur, and the gas provided is the residual nitrogen of the air, together with about 15 per cent of sulphurous acid and a good deal of sulphuric acid in suspension. The process is most effective in destroying rats, mice and cockroaches in cabins and empty holds of the ship, but there is no evidence as yet that it is equally effective in holds which are full of cargo, and the treatment recommended of about one hour for each hold is quite insufficient to destroy vermin. There is no risk of fire or explosion, nor of asphyxiation, because the gas is so unpleasant that everyone would receive due warning of its approach, and, unlike carbonic oxide, it kills insects. The disadvantages of the process are: that it causes serious damage to food, such as fruit, flour or meat, and slight damage to metal work, that it is absorbed to a great extent by articles of cargo, and therefore penetrates them very slowly, and it is at present very expensive, the charge for disinfecting a vessel of 3000 tons being £44.

Burning sulphur is not applicable to holds full of cargo, but may be used in cabins and empty holds, the proportion of

sulphurous acid is about two or three per cent in the air, which is amply sufficient to destroy all animals. At least one and a half pounds of sulphur per 1000 cubic feet of air space are needed for each hold.

Liquid sulphurous acid can be employed for both full and empty holds, it is more expensive than sulphur, but causes less damage than this process or the Clayton gas.

Carbonic oxide gas is used for destroying rats on loaded vessels at Hamburg. It can be very easily and cheaply made, is not absorbed by the cargo and causes no damage to it. On the other hand, it has several disadvantages, it does not disinfect and does not kill insects, it is dangerous to man, it is capable of forming an explosive mixture with air, and it is rather lighter than air and therefore does not pass downwards by gravitation, like the Clayton gas.

Carbonic acid has been used for killing rats on ships at Marseilles, and has the following advantages: it does not damage cargo, it is heavier than air, and it is less dangerous to man than carbonic oxide, because it extinguishes lights before it is dangerous to life. Its disadvantages are: that almost 30 per cent of it is required to kill rats and mice, so that an enormous amount has to be used for a ship, and the expense falls heavily; also, it is not a disinfectant, and it does not always kill insects.

Plague in Mice.—I have already mentioned several historic instances of mice being affected, and, as a matter of fact, experiments show that the degree of susceptibility to plague in the mouse does not fall far short of that of the rat. It is probable that the mouse is generally infected by its stomach, but here again we want information as to whether rat fleas are also parasites of the mouse. Mouse plague has been reported from Formosa and Hongkong in 1896, from Sydney and from Alexandria, where Gotschlich found human plague existing especially in the areas affected with the mouse epizoötic. Many mice have been found by Hunter to be infected by plague bacilli.

Plague in cats.—The importance of the cat as a domestic pet, which frequently devours plague rats, makes it surprising that so little attention has been paid to the possibility of this form

of dissemination of the disease. Many of the older writers have, however, recorded that cats died during an epidemic, and were a danger to human beings. In 1904 Gotschlich found plague cervical buboes in cats in Alexandria, and plague infected cats have been discovered at Sydney and Hongkong, the infection having, apparently, been contracted by the stomach. Hunter reports plague mortality among cats, apparently caught by eating infected rats. The cats suffered from loss of appetite, diarrhœa, vomiting, ruffled hair, emaciation, distended and tender abdomen, and they died in two to six days. At the post-mortems he found great congestion of all the organs and glands, with hæmorrhages of varying size and bubonic swellings about the neck and the mesentery. In the ileum were many small petechiæ, which were well seen shining through the wall of the distended gut. Plague bacilli were found in the fæces and urine, and scattered throughout the body, but chiefly in the lymphatic system.

Plague in other animals.—The question of the existence of plague among domestic animals is especially important in Oriental countries, where so many of them live under the same roof as their owners. Monkeys and rodents (bandicoots, squirrels, ferrets and marmots) acquire plague by natural means; more than one child in India has died of plague after being bitten by an infected squirrel. The mongoose can be inoculated with plague, but does not contract it by the stomach. Guinea pigs and rabbits are readily infected artificially. The carnivora are not very susceptible and, fortunately, dogs do not take it as easily as cats. Horses and the ungulata (calves, sheep and pigs) are slightly susceptible to artificial infection. Pigeons and domestic poultry (hens, turkeys, geese and ducks) have all been found affected in the market place. Bats were found in China to die during an epidemic, and it is stated that bacilli were seen in their corpses.

The case of animals and poultry, which are used for daily consumption, is of great importance. Most small steamers carry live stock for their own consumption and there is nothing to prevent some of them infecting the rats on board, and thus carrying the infection to new countries though no human being need be contaminated.

Among insects, flies die of plague, and bacilli have been found in the bodies or in the excreta of ants, fleas and bugs.

Plague in caged animals.—An unique case of plague infection in a Zoological Garden at Sydney occurred during the epidemic between April and June, 1902. Forty five animals which died in the Gardens, were examined pathologically and bacteriologically; eleven of them were proved to have died of plague, and several others were strongly suspected of this cause of death, though, for various reasons, the bacilli could not be found. Of the eleven animals, seven were marsupials (wallabies and kangaroos), three were guinea pigs, and one was an Indian antelope. None of the birds in the Gardens showed any signs of plague, but 76 rats were also examined and 19 were found to have plague. Also, one of the keepers, belonging to a staff of six, died of plague. The cause of infection was not discovered, but it is possible that the Gardens may have been infected through imported fodder; the nearest house of a plague patient was 330 yards away, and from this house dead rats were removed. The picking out of the marsupials in this outbreak is interesting because they are thought to be zoologically akin to the rodents. The post mortem appearances of the animals were: general congestion, œdema and minute hæmorrhages into various viscera and under the skin, enlargement of the spleen and lymphatic glands, and sometimes pneumonia.

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BILHARZIOSIS.

Synonyms.—Bilharzia disease; endemic hæmaturia. The worm is called by various writers, Distomum hæmatobium, Bilharzia hæmatobia, Distoma capense, or by its original name, Schistosomum hæmatobium.

Definition.—A chronic, endemic human disease of many tropical countries, characterized by hæmaturia, cystitis, rectal irritation and other symptoms, caused by the deposit in the tissues of countless eggs laid by the *Schistosomum hæmatobium* worm. In advanced cases there are many serious complications, which may eventually cause the death of the patient.

History.—Since quite ancient times the natives of Egypt have suffered greatly from urinary calculi, possibly partly due to this disease. The French troops suffered much from hæmaturia in 1799—1801, during their campaign in Egypt, but never discovered the cause. In 1851, Dr Bilharz at Kasr-el-Ainy hospital first found that hæmaturia was occasioned by the worm, which has been named after him, and in 1864, Dr. John Harley discovered this parasite in cases from South Africa. Bilharz and Griesinger found the disease in Cairo in 117 out of 363 autopsies, or 32 per cent. Sonsino performed 91 post mortem examinations in Lower Egypt between 1875 and 1883, and found bilharzial infection in 42 (46 per cent). Kaufmann, in 1893, had performed 500 autopsies in Cairo, in 165 of which, or 33 per cent, bilharzia was present. His figures included 369 males, with 40 per cent infection, and 131 females with 11·5 per cent attacked.

Distribution.—In Europe the disease is nowhere endemic, but individuals often bring it home with them from Egypt, South Africa and other places. Curiously enough, Berkeley Hill, however, described a case of bilharzia in an English woman who had lived all her life at Erith or Gravesend.

In Asia it has been reported from Mecca, the Hedjaz, Arabia, Syria, Mesopotamia in the valleys of the Tigris and Euphrates, Persia and the west coast of India. It is known in Mauritius and single cases have lately been reported from Cyprus, America (Illinois) and the West Indies. These sporadic cases, occurring in countries where the disease is not known to exist, are probably due to infection from some bilharzial visitor.

But it is in Africa that it is best known and most widely distributed. In Lower Egypt the worm is found in at least one-third of all the autopsies, (38 per cent out of 261 post mortems at Kasr el Ainy during 1904); and in 1901 (an average year) there were treated at Kasr el Ainy 930 cases, most of which came from Lower Egypt. In the Sudan it is common amongst imported Egyptians, but it was thought not to be endemic until Dr. Balfour recently found it in three boys who had never been out of the Sudan. Sporadic cases have been reported in Arabs from Kassala, Darfur and Kordofan, and the disease is endemic in Tunis and Algiers. On the west coast, it is known in Nigeria, the German Cameroons and Angola; and on the east coast, all the way from Egypt to the Cape, including Zanzibar and Abyssinia, where the Italian troops suffered considerably from it during their campaign.

In Central Africa it is endemic in Uganda, British Central Africa and the Congo State, while in South Africa it is well known in Delagoa Bay, Port Elizabeth, Natal, Bloemfontein, the Transvaal and Kaffraria. The disease is less serious in South Africa than in Egypt.

It is remarkable that bilharziosis is one of the chief diseases of Africa and yet is so little distributed in Asia, where the conditions of climate and human life are so similar.

Of 621 cases analysed by Mr F. Milton, 121 hospital patients gave Cairo as their temporary address, while, of the remaining 500, no less than 82 per cent came from Lower Egypt, and only 18 per cent from Upper Egypt. The provinces most affected are: Sharqia, Qaliubia, Menufia, Daqahlia and Giza, whilst the least affected are Girga, Kena and Assiut. Very many cases are present in the Egyptian troops, and occasionally cases are met with in hospitals in Great Britain, of men who, apparently, contracted the disease whilst serving in the army in Egypt.

NATURAL HISTORY.

The three pages which follow are taken, by his permission, from Professor Looss's article in Mense's "Handbuch der Tropen Krankheiten."

The *Schistosomum hæmatobium* belongs to the small group of two sexed trematodes and appears to the naked eye milk white, round, pointed at both ends, and about one cm. in length. Under the microscope, two suckers are to be seen at one end, one behind the other. The body of the male is closely covered with small tubercles and behind the second sucker is the beginning of the gynæcophoric canal, which is formed by the doubling in of the lateral parts of the worm, like the lappets of an overcoat. The female is much longer than the male, filiform and only the anterior half of her body is white, her posterior half is grey, and traversed all along its axis by a dark brown stripe in zigzag, which represents the intestinal canal filled with blood.

The sexes live apart while young, but as soon as maturity is reached the female enters the gynæcophoric canal of the male, out of which part of her body often protrudes, and yet she is sometimes able to hide herself so completely that she can only be discovered by the aid of a microscope. The principal *habitat* is in the portal vein. The worms found in the liver are always young, and when mated pairs are seen in the portal vein, the female has no normal eggs in her uterus, and no great number of them. Bilharz originally found in the intestinal veins and in the wall of the bladder coupled worms, and in the bladder the females contained plenty of mature eggs. The veins of the pelvis must therefore be regarded as the habitual home of the mature worms. The males, carrying the females with them, reach the submucous tissue of the bladder and rectum by means of their strong muscles and very energetic movements, and, in a well marked case, these organs are so rich in worms, that a pair may be found in every area of half a cm. square.

Development.—The eggs are deposited in the veins; they are somewhat spindle shaped and measure on an average 0·08 by 0·03 m.m. After their entry into the tissues, segmentation takes place, and the eggs grow until they are usually 0·11 to 0·12 m.m.

long and 0·04 to 0·05 m.m. wide. Their thin yellow shell has at its posterior end a small pointed spine, but often another form of egg is seen with a considerably larger lateral spine. The fact that lateral-spined eggs are often found in the fæces of Bilharzia cases and very seldom in the urine, has led to the belief that the different shaped eggs might possibly belong to two species of Schistosomum, but Professor Looss is not in favour of this view and rather believes that the eggs with the lateral spines are abnormally formed specimens. Like Bilharz, he found them chiefly in isolated females and thinks it possible that it is only immature females who are able to lay eggs with lateral spines.

The eggs are doubtless expelled from the body of the female before the worms reach the capillaries, that is to say, during their journey from the portal vein to the pelvic organs; these eggs are carried along by the blood, and may remain in some organ such as the liver.

When once the worms have reached the capillaries most of the eggs are held fast to the spot where they were deposited by the pressure of the surrounding tissues. Bilharziosis like so many other diseases is thus originally a local manifestation. (H. Milton).

The eggs, squeezed together by the contraction of the muscles of the bladder and intestines, penetrate into the tissues, and travel along like any other foreign body until they reach the surface and are expelled. A strong lateral spine hinders this progress, for such eggs are like a boat with a misplaced rudder and instead of proceeding in a straight line, are obliged to move more or less in a circle, and thus collect in the tissues, especially in the bladder, for in the rectum they are probably dislodged by the fæces.

Many eggs die during their process of development, and vary in size according to their growth at the time of their death, but when found still alive in the urine or fæces of a patient they generally contain a ripe *miracidium*. Water is evidently the essential for the life of the miracidium, it emerges from the egg in a few minutes in warm weather and swims rapidly about by means of its cilia. But no one has yet succeeded in keeping it alive for longer than 30—40 hours, and so far experiments

made to try and transfer it to animals which might possibly act as intermediary hosts have invariably failed.

Infection of man. Two possibilities have been suggested, transmission by means of the stomach or by the skin. By the analogy of other trematodes, it has been supposed by most observers that the miracidium must enter an animal as yet undiscovered, get to the human stomach with unfiltered drinking water, and so develop into bilharzia worms. Bilharziosis is so widely spread in Egypt that it is fairly obvious that the intermediary host cannot be any rare creature.

Looss has for some years been persuaded that the miracidium enters directly into man, and he excludes the possibility of entry by the mouth in drinking water, because the miracidia cannot live for one minute in a solution of 1/1000 hydrochloric acid, nor for three minutes in a solution of 1/2000. How then is it possible for them to traverse the human stomach alive? There is no evidence that entry can occur by the urethra or rectum. There remains for discussion only the mode of entrance by the skin, which was long ago suggested as the possible explanation of the fact that bathers were specially attacked by the disease, and as men and boys bathe in South Africa more than girls it was thought by Harley, Brock and others that this difference in their habits might account for the comparative immunity of the female sex. As regards the prevalence of the disease in Egypt, Dr. Talaat, who has specially examined the population of some country districts, finds that in the poorest classes, where men and women work together in the fields, both sexes suffer equally from bilharziosis. Among those who are less poor, it is only the men who work on the land, and the disease is rare among the women, while among those who are well-to-do, even the men are not engaged in agricultural labour, and bilharziosis seldom occurs among them. Now for all these three classes and for both sexes the drinking water is the same, unfiltered from the Nile. Goebel also states that in towns it is especially earth workers who suffer from the disease. Engel found 35·5 per cent of Cairo street sweepers affected, though there were only 11 doubtful cases among 400 pensioners examined by him in Cairo.

Those who maintain that this miracidium is incapable by its

formation of entering the human skin, may be reminded that the miracidia of other trematodes succeed in penetrating the skin of molluses and then reach the liver of the latter. Having once entered the human body the Bilharzia miracidium must somehow get to the liver, for this is the only organ where very young worms are found, and it seems clear that they disperse from the liver to other parts of the body. When they have reached maturity, the worms enter the large branches of the portal vein, join sexually and travel on to the veins of the pelvic organs to lay their eggs there. Some coupled pairs probably go astray on the way and lay their eggs, thus accounting for unusual and isolated Bilharzia tumours..

For the theory of infection by the skin, it is only necessary that mud or water fouled by living eggs should remain for a little while in contact with the skin. In Egypt, the middle-class and poor children of both town and country districts become affected because they play in small dirty ponds where infection is easy if only one of them discharges Bilharzia eggs into the water. But among adults it is chiefly the country dwellers who suffer, partly because the town children as they grow older cease to paddle in the ponds, and therefore their chances of re-infection become less and their hæmaturia slowly disappears. The country children, on the other hand, develop into peasants, who work barefooted in groups on the wet land where re-infection may constantly occur.

The following case must, I think, have been infected by bathing in Nile water. A rich Englishman had never been out of Europe until the winter he spent in Egypt. He lived on a Nile boat; and having traumatic disease of the spine was seldom carried on land. He was a gouty individual, and had been lying on his back for eight years. He never drank Nile water, unless it had been boiled, his habitual beverage being imported Malvern water. He never bathed in the river, but sometimes had two baths of Nile water during the day. His urine, two years after he left Egypt, was carefully examined, because he had then developed a renal calculus, and several bilharzia eggs were found, but he had no hæmaturia or other symptoms of bilharziosis.

Zoology.—Very little is known about the zoological distribu-

tion of this parasite. Cobbold found a schistosomum in a sooty monkey (Cercopithecus fuliginosus) from Africa, which died in London in 1857, and he described it as bilharzia magna, but this is now believed to have been bilharzia hæmatobia

Sonsino, in 1876, discovered a similar trematode (Schistosomum bovis) in the portal vein of a three-year-old bull, in the slaughter house at Zagazig; he found 35 living worms, the females lying in the male gynæcophoric canal, and appearing like a thread of black silk at each extremity. The males were somewhat broader than the human species, and the eggs were spindle-shaped and contained no embryos.

In 1904 Dr. Catto, in making an autopsy upon the body of a Chinaman, who had died of cholera in Singapore, had occasion to microscopically examine a fibrous growth in the neighbourhood of the appendix. He noticed a large number of eggs, which were something like those of the bilharzia worm, but were unprovided with spines. Further research into the tissues of the alimentary system led to the discovery of an adult parasite, which was recognized as a schistosomum, but different to the African variety, for the surface of its body is smooth, being totally devoid of the little tubercles which are so distinctive a sign of the Schistosomum hæmatobium. In the post mortem of this case the liver was found to be enlarged and nodular, the spleen greatly enlarged, showing malarial pigmentation, the mesentery thickened, the large intestine and posterior wall of the bladder thickened, the mesenteric and many intrapelvic glands were greatly increased in size, the largest assuming the dimensions of a small hen's egg. Microscopic slides of these tissues showed small, oval, double-walled bodies, from 68 to 70 μ. in length by 34 to 40 μ. in breadth. The ova existed in prodigious numbers in the submucous membrane of the large intestine and in smaller numbers in the subperitoneal tissues of the large intestine.

It now appears that Katsurada, in Japan, found this parasite in April 1904 and named it *Schistosomum Japonicum*.

He had the opportunity of studying 12 cases in one district, and in the fæces of five of them, he found the eggs; he also found eggs in three livers and one large intestine, but never in the bladder. In two cats belonging to the same district, he

found in one, eggs in the liver, and in the second, many worms in the portal and mesenteric veins, besides eggs in the liver and the walls of the large intestine.

CAUSES.

Age.—The disease is unknown in infants, but may be found in males of any age from 4 to 70. Three-fourths of the Cairo cases which come to hospital are between 15 and 45 years of age.

Sex.—It is rare in females, except among little girls and peasant women who, like the men, work bare-legged on the flooded ploughed land.

In investigating this point Mr. F. Milton found that out of a total of 1346 hospital patients in Cairo, over 94 per cent were males, but that the relative proportion of cases under puberty was nine males to 1 female, and that after the age of puberty there were 20 males to one female. This striking difference in ratio can be explained by remembering that the children of both sexes in the country districts lead a very similar life, playing together in the irrigated fields and muddy pools, but when the girls come to a marriageable age they are much more confined to the house, or only work on dry fields. They are thus preserved from the re-infection which attacks males. In 1904 an Egyptian girl died of tuberculosis at Kasr el Ainy and was found to be infected by Bilharzia. She said she was 10 years old, but she looked not more than seven.

Races.—All are liable, but Europeans rarely contract the disease, excepting a few Greeks who lead similar lives to the Egyptians. Of the Kasr-el-Ainy patients 90 per cent are Egyptian Muhammedans, the remaining ten per cent being made up of Sudanese, Berberines, Copts, Greeks etc. The three last named races are mostly employed in indoor work, which accounts for the small number attacked. The Sudanese work in the fields, and therefore ought not to escape, but it is possible that they may enjoy some degree of freedom, which is certainly the case with them in Ankylostomiasis and some other diseases. It is possible even that they may be protected by their habits of oiling the skin. In South Africa the Colonial and Dutch boys, who paddle and bathe, are by no means

exempt, but the disease is extremely rare there in girls, who do not disport themselves in water.

Occupation.—In the towns the patients are chiefly children, or adults employed as road labourers; for the country districts I must again quote from Mr. F. Milton's statistics. Of 650 male adults, whose occupation was known, 71 per cent were peasants and seven per cent were itinerant teachers (fiki), attached to Mosque schools, but themselves belonging to the peasant class. Most of the remainder consisted of policemen, servants, labourers, village barbers and gardeners, all springing from the peasant class and certain to have spent their boyhood in bathing and paddling in dirty pools and streams which irrigate the land. In order to try and discover what proportion of the male population suffer from bilharziosis, the urine of 100 consecutive patients, admitted for all diseases to the medical wards of Kasr-el-Ainy in June 1902, was specially examined by microscope; no less than 35 were found to have bilharzia eggs, although only two patients complained of any symptoms of the disease. (F. Milton).

Dr. Kautsky, in examining a school of 124 Egyptian boys, near Cairo, found that 79 per cent had bilharzia eggs in their urine, though they all professed to be perfectly well.

Climate and Season.—The land in Lower Egypt is flooded during the late summer and autumn and probably infection occurs during the early winter months; after an incubation period of about three months, the patients arrive for treatment in the summer and autumn.

If the chief infection of bilharziosis is by the skin, it is worth while emphasizing the fact that the provinces of Lower Egypt, which furnish the bulk of cases seen in Cairo, are supplied with water by irrigation canals practically for all the year, whereas the peasants of Upper Egypt, where basin irrigation still exists, get water for their crops only at the time of the Nile flood, and, therefore, have a very much shorter period of time during which possible infection may take place. The disease is probably limited to warm countries, because the miracidium so obviously prefers warmth. Chronic cases void more eggs in the urine in spring than at other times of the year.

Varieties of the disease.—The Kasr-el-Ainy statistics for 1901,

which include the whole of the in-patients and out-patients treated for this disease in the hospital, numbering 930, provide us wtih the following percentages: Bilharzia affecting the bladder 64 per cent, bilharzia of the bladder complicated by vesical calculus 14 per cent, urinary fistulæ 14 per cent, bilharzia of the rectum and general bilharziosis 5 per cent, and the remaining 5 per cent were cases in which the patients died of other diseases, but bilharzia worms were found in their portal system.

SYMPTOMS.

The incubation is doubtful, but apparently varies from two to four months. The parasite may certainly be present in the human body for six or more months without producing any symptoms. Hæmaturia is the most important early sign; the patient finds his shirt stained, and a few drops of blood are passed at the end of urination. This hæmorrhage, which is almost diagnostic of the disease, is caused by the contracting bladder bursting some of the blood-vessels filled with eggs. At an early stage of the disease the urine, voided before the bladder seriously contracts, is quite normal, but if the after-coming blood be examined, or if the whole of the liquid be centrifugalized or allowed to stand in a conical vessel, the microscope will reveal red blood-cells, leucocytes, epithelial cells, and bilharzia eggs entangled in mucus. The hæmaturia is increased after any excess of eating, alcoholic drinking, or exertion; and many patients have first noticed it after an injury, such as a fall from a horse. The individual finds himself obliged to urinate more frequently than before, and though there is no pain he has a slight pricking at the prepuce or at the root of the penis. Gradually he develops a little back-ache and general lassitude, but the symptoms are indistinct, and so common in bilharzia countries that natives never dream of obtaining medical advice for them.

The adult parasites would be harmless in themselves, if it were not that each female deposits many thousands of eggs, and these eggs, in process of elimination, produce great discomfort, and sometimes very serious illness. The eggs, if deposited in the mucous membrane of the bladder produce

hæmaturia, but if in the rectum, the chief symptoms will be mistaken by the patient for piles or dysentery. If the urine, with or without blood, which is passed at the end of micturition, is received in a urine glass, there will often be seen colourless or white filaments about one inch long, or small, opaque, roundish masses, which quickly settle, and these will probably be found to contain eggs. The number of ova in a typical case which are passed every day must reach several thousands.

An intelligent patient can generally tell you of some pain and discomfort, though he can not always locate it, this will sometimes be felt in the buttocks, front of the thighs or perineum, and when the disease becomes advanced there will generally be pain in the inguinal, hypogastric and sacral regions.

The *bladder* is the organ most often affected, the symptoms being those of subacute catarrh, increased frequency of micturition, a feeling of not having emptied the bladder, scalding and pain in the perineum and in the lumbar region. Cystitis occurs when the mucous membrane of the bladder has become thickened by the deposit of eggs in raised sandy patches, with early fibrosis.

Septic cystitis is the result of germs which have been introduced, with or without instruments, from the urethra into the bladder, for when once the bilharzia patches have become large, there is no complete contraction of the bladder, and therefore a constant dribbling of urine exists. In the final stages numerous papillomata in the bladder, with sloughing from their ulcerated surfaces, cause severe tenesmus, so that the patient can only get relief by grasping the root of his penis. An enlarged prostate adds to all his troubles.

When eggs have been deposited in the *rectum*, there is an increased secretion of mucus and some straining, followed by papillomata causing relaxation of the sphincters and prolapse of mucous membrane. When the mucous membrane near the sphincters is infiltrated, symptoms very like dysentery are produced, and may easily deceive those unacquainted with the two diseases. In the worst cases there are huge polypi formed, with ulcerating sloughs, and the patient, as when the

bladder is similarly implicated, dies exhausted by pain and constant tenesmus.

The *vagina* is the part most affected in the female sex, the symptoms being chiefly those of subacute vaginitis, due to thickening of the mucous membrane, especially on the posterior wall.

No symptoms are known to show when the liver and lungs are affected, but to the various forms of cirrhosis there must be added a bilharzial cirrhosis of the liver, of which I have seen several cases. Some hospital patients have suffered from ascites, for which there was no discoverable cause, but there was evidence of bilharzial growth in the rectum. Many such cases have left the hospital alive, and therefore there was no absolute pathological certainty of the absence of other causes for the ascites, but some few have been kept in the hospital until they died, and at the post mortem there has not only been no evidence of cancer, syphilis, alcohol, malaria or Banti's splenic anæmia, but there has been a distinct peri-portal cirrhosis of the bilharzial type, and in the rectum there were, invariably, bilharzial papillomata. It must, therefore, be accepted in Egypt that bilharzial cirrhosis may, sometimes, produce ascites.

The *anæmia* is slight, even after the symptoms have existed several years, so that patients do not come to the hospital complaining of anæmic symptoms like those suffering from ankylostomiasis, but they are driven there by burning pain in the perineum and urethra, hæmorrhage after urination, or blood and mucous discharge from the rectum. In one peasant, who had had all these symptoms for three years in the country, I found 55 bilharzia eggs on one slide, taken from the deposit of his urine, yet he had no apparent anæmia. However, the blood of bilharzia patients is found to flow slowly; there is a slight diminution of hæmoglobin, say 84 per cent, with a slight decrease of the red cells, perhaps to four millions. The small amount of leucocytosis present is probably caused by the complications. There is nearly always an increased number of eosinophiles, the average of many cases being 16 per cent., sometimes rising to 40 or even 52 per cent. (Kautsky). The lowest record which I know was 1·3 per cent.

MORTALITY.

We have not enough statistics to be certain of the death-rate, but Goebel finds that out of 1,684 cases treated in the Alexandria Deaconesses' Hospital, 5·8 per cent died. Of course it is only the worst and most advanced cases which ever come to the hospitals, but on the other hand many hopeless patients leave the hospitals in order to die at home.

COMPLICATIONS.

The hæmorrhage in the bladder may produce a solid clot which blocks up the whole viscus, so that a catheter draws nothing off; and if the condition be not relieved by further operation, the patient gets acute retention of urine and rapidly dies of uræmia. The thickened bladder may be so distinct that it can be felt like a small cricket-ball above the pubes, while in women the tumour of the vagina or about the urethra may be so large as to be confounded with cancer.

Calculi in the kidney and more especially in the bladder are the best known and most important sequelæ, for in all countries where bilharziosis is prevalent stones are common. To Kasr-el-Ainy hospital alone there come every year about 150 patients suffering from stone, and in all of them bilharzia eggs are either still present in the urine, or there is a history of recent evidence of the parasite. The majority of these stones have been formed in the bladder, not in the kidney. Only a limited number of stones have been examined microscopically, and I think that, at present, the question must be left undecided whether it is true or not that the bilharzia eggs actually form a nucleus of some of the calculi.

The stone-symptoms are masked by the cystitis caused by the bilharzia, but the stone greatly aggravates the patient's sufferings, and there is a corresponding relief when lithotrity has been performed. In the later stages of the disease the stones are formed of phosphates, and being soft, are not so painful, and are sometimes difficult to diagnose from patches of rough mucous membrane of the bladder already encrusted with phosphatic deposit. The great majority of stone-patients come

from Lower Egypt, very few from Upper Egypt or the Sudan. Enormous stones, weighing from 20 to 35 ounces have been successfully removed by Mr. H. Milton and other surgeons during the last 20 years, but now there are so many operators in Egypt, that very large stones have become rare. Mr. Madden operated upon 100 cases in 1902, varying in weight from 4 grains to 7 ounces, but only five of his hospital calculi weighed more than 4 ounces; only one of the patients was a female, and the youngest case was a child under two years of age. It is impossible to judge the size of the stone by symptoms alone, for sometimes a tiny calculus lodged near the neck of the bladder will produce very severe pain and irritability, while a stone completely filling the bladder may not cause much inconvenience.

Urinary fistulæ are another very serious complication among men, originating in the roof or the floor of the urethra. In the roof-cases the symptoms are mild and will probably be masked by previous bladder-disease, until a subcutaneous abscess forms in the perineum, from which there discharges a small amount of unhealthy pus, containing bilharzia eggs. The fistula has now formed, and makes no attempt to heal; it may be recognised by one or more sinuses in the perineum, or the posterior part of the scrotum, or even on the pubes or the thighs, but always away from the direct line of the urethra.

Fistulæ arising from the floor of the urethra are much more important; the opening is generally in the perineum, but it may be in the penis in the front of the scrotum, and is always in the immediate line of the urethra. The skin is extensively adherent and thickened, and becomes a sharply defined mass of stony hardness, connected with the urethra, the meatus being narrow and scarred, with, perhaps, thin, unhealthy pus oozing from it, and when the catheter is passed a stricture is discovered. Many patients do not come to hospital until an ulcer has formed, followed by a peri-urethral abscess and a stricture. During the years 1890—1901 there were 1684 cases of bilharzia treated in the Deaconness's hospital in Alexandria, and it was found that 18·4 per cent of them had developed perineal fistulæ.

CONCURRENT DISEASES.

Cancer is said to develop sometimes from the irritative overgrowth of the glands in the neighbourhood of the bladder, but statistics on this subject are still wanting; in my experience it is distinctly rare. In Egypt the most common accompaniments of bilharziosis are pellagra and ankylostomiasis, and to a lesser extent favus of the head, while, as in all infected countries, there is a great liability to many of the other entozoa.

MORBID ANATOMY.

At an autopsy the infiltration of the ova can be best seen in the urethra, bladder, ureters, rectum and liver. In the *urethra* there may be small, sandy patches, due to deposited eggs followed by fibrosis, but the most common lesion will be fistula. As regards origin, there is only one springing from the floor to 20 arising in the roof or the pubic side of the urethra, and this complication is so common that there are always numbers among the out-patients waiting for admission to the crowded hospitals of Cairo and Alexandria.

It is quite possible to meet with a bilharzial lesion in the glans penis or in the perineum, which may surround the urethra without the urethra itself being primarily affected.

A perineal fistula may give rise to a stricture, but in the more common variety of fistula there is no stricture, though the urethra may be made into a rigid, thickened, twisted tube.

In the *bladder* the first change visible to the naked eye is small pellucid bodies in the mucous membrane, raising its surface above the surrounding level; these bodies gradually coalesce until a part or the whole of the bladder is affected, the earliest place being the trigone. As the eggs increase in number, they are gradually enveloped in fibrous tissue like other foreign bodies, and soon cause blocking of capillaries. Soon the sandy patches, yellowish-grey or yellowish-brown in colour, can be felt by the operator's sound; and, owing to the deficiency of the blood supply, these patches gradually lose their vitality, break down and slough, causing ulcers and crevices, which retain the urine and set up decomposition. When the hypertrophy of the

mucous membrane overbalances the formation of new fibrous tissue, papillomata and polypoid growths of great vascularity, and even solid tumours are formed.

At the post mortem of a case where the disease in the bladder is at its commencement, we may find one or two small patches, where the mucous membrane is rough, slightly raised and of a peculiar pinkish, yellow colour; a scraping of this sandy surface will show, under the microscope, a large number of eggs. In other cases there are several small discrete vesicles, not larger in size than a pin's head, containing a whitish fluid, in which many eggs can be found. But in these early cases it is often impossible to find any worms in the portal blood. One may find that about half of the bladder mucous membrane is covered with a yellowish, pigmented, leathery thickening, which looks as if it had been preserved in spirit or may resemble finely granular sandstone. This causes the knife to make a grating sound on section, and under the microscope the eggs may have no living contents, being partly filled with carbonate of lime. This condition of things is quite different to the other variety of single or multiple excrescences of a reddish-yellow colour, which are covered with an incrustation consisting of phosphates and the eggs of the parasite. In this latter stage it is, apparently, the living animals which act as an irritant on the mucous membrane and the sub-mucous tissue, whereas, in the earlier stage, the pathological appearances are produced by the ova. The following is an example of advanced and dangerous disease in the bladder: A fellah, aged 25, had suffered for many years from hæmaturia and bladder symptoms, and for two years from signs of vesical calculus. When he was admitted into the Government hospital at Alexandria under Schiess Bey he was found to be too ill to undergo operation, and he died of uræmia shortly afterwards. At the post mortem, twelve hours later, his heart was hypertrophied, there were no distomas in the cardiac blood, the mitral valve was thick, and on the aortic valve there were three atheromatous deposits each about the size of a lentil. The liver was small and congested, and the portal vein contained thick, dark-red blood with 30 distomas in one tea-spoonful. The mesenteric glands were large, the stomach, intestines, spleen and lungs were normal.

The kidneys were large, showing several cysts, and contained ammoniacal urine, while the pelvis and calyces of both kidneys were greatly dilated. The ureters were thickened and so dilated as to measure, in the collapsed condition, 1.5 cm., but their mucous membrane was intact. The bladder was 3 cm. thick, and in its cavity there was a calculus the size of a nut, besides two small calculi buried in the thickened wall. A fourth calculus, at the lower end of the left ureter, was afterwards discovered by Mr Shattock, who described the bladder as follows: "About its neck the mucous and submucous coats are thickened and indurated from chronic inflammation; and in this situation the mucous membrane of the posterior wall presents five or more small, but deep ulcers, two of them considerably undermined. The inflammatory process involves the tissues contiguous with the base of the bladder for a wide extent, the vesicula seminalis and lower end of the ureter being firmly incorporated with the parts in front by intervening indurated fat and connective tissue."

On section, many eggs were found in the bladder and ureters. In the *ureter* there occur appearances very similar to those I have described in the bladder. The changes are seen as sandy patches at several spots, but most commonly low down, where the ureter opens into the bladder. There are sometimes annular deposits, which often so narrow the calibre of the duct that the finest sound can hardly be passed through; the natural consequence of such a constriction is a dilatation of the upper two thirds of the ureter, and then of the pelvis and calyces of the kidney.

Changes in the *kidney* depend chiefly upon the amount of obstruction to the flow of urine; the pelvis gradually stretches until hydronephrosis or pyonephrosis may be caused, and several times the kidney has been converted literally into a thin bag of pus. Eggs are seldom found in the kidney, but have been seen in the seminal vesicles and prostate.

The *rectum* is surrounded by a large plexus of veins, communicating with the portal system, most of which have no valves, and it is, therefore, an ideal place for the female worms to deposit their eggs. The ordinary case shows a general thickening of the mucous membrane, which is found to be unnaturally red and of a fine, granular appearance, it bleeds readily, there

is an excessive secretion of mucus, and the sphincter is somewhat relaxed. There may be, as in other bilharzia lesions, some destruction of the normal tissue, and formation of fibrous tissue in its place, but in advanced rectal cases there are polypoid growths which, to a greater or less extent, may not only appear in the rectum itself, but extend upwards throughout the whole length of the great intestine, and even the lower six inches of the small intestine. Stricture of the rectum never seems to occur, and fistula in ano is quite rare.

Bilharzial cirrhosis of the *liver* has long been recognized in Egypt, but the best pathological account of it is that of Dr. Symmers.

The liver is generally enlarged, weighing more than 60 ounces. To the naked eye its surface presents a peculiar appearance, due to white markings of increased connective tissue appearing through the capsule, and a number of perihepatic nodules projecting from the capsule, of which they are distinct growths. On section, the liver shows an enormous increase of new white fibrous tissue, "so that its cut surface looks as if a number of white clay pipe-stems had been thrust at various angles through it." The parenchyma of the organ is homogeneous in appearance, and has a peculiar drab colour, which contrasts very markedly with the pinkish-white of the new fibrous tissue. There is no prominence of liver lobules, nor retraction of the capsule, as seen in other forms of liver cirrhosis. Microscopically, there is a great increase of the periportal connective tissue of the sublobular and larger portal canals, the fibres of which are mostly wavy and parallel, but here and there arranged concentrically and surrounding ova. The eggs found in the liver consist usually only of the shell with lateral spines; no eggs are seen lying free among the hepatic cells, nor any, usually, in the larger blood-vessels which permeate the new cirrhotic tissue. The whitish nodules on the surface of the liver consist of fibrous tissue containing eggs, and similar fibrous overgrowths are occasionally found in other parts of the body, where the new tissue also contains eggs.

The *portal vein* should always be opened to see if it contains any worms; sometimes only two or three will be found, at other times each teaspoonful of blood may contain 30 parasites,

and in one of my cases 436 worms were found in blood from the liver. In order to make a thorough examination it is best to slit open the vein by longitudinal incision, and scoop out the blood with a teaspoon; then to cut the liver into pieces and wash these in saline solution while applying some pressure with the hands. The worms can be easily seen by receiving the blood in a glass dish or a black tray. They may also be found in the inferior vena cava, the common iliac, and some other veins.

It is rare to find any eggs in the *lung*, for apparently the worms only migrate there when they have lost their way; and though I have often given expectorants to bilharzia patients to increase their sputa, we have never succeeded in finding any eggs there. Eggs have been discovered in the skin, but only in warts on the perineum; also in the mesenteric glands. It used to be thought that they were never found in the spleen, but Dr. Symmers has shown them in fibrous nodules, the size of a split pea, on the outside of the spleen.

Among the rarer sites, living worms, coupled, have been found in the polypoid growths of the rectum, descending colon, bladder, in outgrowths from the intestinal peritoneum and, in December 1904, by Symmers, in the blood of the lung. Griesinger found eggs and empty egg shells in the blood inside the heart, Goebel found them in the pancreas of a fellah, who died of cancer of the bladder and bilharziosis, and also in the stomach contents of a boy aged 12 who had a gastric fistula, following tubercular peritonitis. Mr. F. Milton found them in a case of multiple superficial sinuses in the sacral region of a boy aged 12, who had, apparently, no signs of bilharzia either in his rectum or bladder. Symmers found, in the post mortem room, a tumour as large as half a walnut, which was attached to the free edge of the broad ligament of a young girl; the tumour, on section, consisted entirely of bilharzia eggs and recent fibrous tissue.

Mr. Madden has reported some interesting cases of bilharzial infection of the transverse colon, peritoneum, omentum and mesenteric glands, a tumour of the abdominal wall, and a series of cases affecting the vagina, vulva and cervix of the uterus.

Dr. Crimp, in 1904, while making post mortem examinations

at Kasr el Ainy, found eggs several times in the neighbourhood of the appendix. In the museum of the Cairo Medical School there is an excellent series of preparations showing bilharzial changes in the bladder, ureter, urethra, rectum, colon, cæcum, ileum, liver, spleen, uterus, broad ligament and vulva.

DIAGNOSIS.

The disease in the urinary passages can be detected by the drops of blood passed at the end of urination, and by the eggs found in the deposit of urine which has been centrifugalized or allowed to stand for a few hours. If no eggs can be found in a suspected case, it may be advisable to take the whole quantity of urine for 24 hours and centrifugalize the deposit from it, or to pass a catheter and examine the few drops of urine remaining in the instrument. This will probably prevent evidence of bladder changes being found post mortem where no eggs had been discovered during life. It must not be forgotten that eggs are sometimes found in the fæces but not in the urine.

The diagnosis between simple cystitis and cystitis complicated by calculus can only be made by examination with a sound. The rectal tenesmus can be distinguished from signs of acute dysentery because in the latter the disease begins suddenly with diarrhœa and generally some fever, while the tongue is coated and there is pain over the colon. Bilharzial cases will show eggs in the blood and mucus passed from the rectum, and on examination with the finger a definite roughness, if not polypoid growths, can be felt. A peri-urethral abscess, arising from a fistula in the floor of the urethra, resembles a stone impacted in the urethra, but in bilharziosis there is a continuous escape of unhealthy pus from the meatus, the meatus itself has a scarred, glazed, narrowed look, and the urethra is very hard to the touch all the way from the swelling to the meatus. In case of doubt a sound must be passed down the urethra. The discharge about the meatus and glans penis has sometimes wrongly been taken for gonorrhœa.

In the case of tumours of the female genital organs, it is better to snip off a piece of thickened mucous membrane, to

search for eggs under the microscope, and so exclude the possibility of cancer.

PROGNOSIS.

The disease is certainly of less severity in South Africa than in North Africa, and one can only suppose that this is due to a greater number of worms being available in the latter than in the former, for the distressing symptoms of the disease seem to depend chiefly on whether there are hundreds of thousands of eggs deposited in the tissues, or only thousands. We know that each female during coupling has the power of laying an enormous number of eggs, and the prognosis therefore depends a great deal upon an unknown factor—that is, how many female worms are existing in the patient's body. Those who are very thoroughly infected die a miserable, painful death from exhaustion and debility, the result of advanced kidney-disease or other complications. But, on the other hand, I know several intelligent Egyptians, more than fifty years of age, who state that they have had bilharziosis since boyhood, without suffering any very ill effects. But as they have lived always in Egypt, one cannot tell how often they may have been re-infected. I have also known two Englishmen who still occasionally passed eggs in their urine for ten years after leaving bilharzial countries; both suffered discomfort, which they put down to the presence of their chronic enemy, but both could do a fair amount of bodily and mental work.

A third Englishman was infected in Africa in January 1877, and believes he is still the unwilling host of the parasite. He tells me that eggs were seen in his urine as late as 1901. But I believe that most cases of ordinary bilharzia infection lose their symptoms within three years after leaving the country in which they contracted the disease.

TREATMENT.

No method is as yet known of killing the worms in the human body. Thymol, which acts so well upon most intestinal parasites, is useless here, even when the ravages seem to be

confined to the rectum and descending colon. The liquid extract of male fern, in doses of 15 minims three times a day, is the only drug of any known value, for it controls or abolishes hæmaturia, allays the irritation in the bladder, and reduces the number of eggs passed in the urine and fæces. But it must not be continued for more than about a fortnight at a time, as the following case will show: A healthy-looking native, aged 24, from Upper Egypt, was admitted to hospital under my care on November 25 for bilharziosis of one year's duration. He denied the existence of hæmaturia, and, unlike most of the peasants from the Delta, he seemed to be unacquainted with the disease; after centrifugalizing his urine, only two or three eggs were seen on each slide. On December 4 he began taking male fern m. xv., thrice daily; and eggs were found every day in his urine until December 11, when they disappeared, and never recurred during the time I kept him in hospital. For some reason his medicine was not discontinued until December 24, when I found, after twenty days of the drug, that he was suffering from giddiness, pain in the head, eye-balls and epigastrium, singing in the ears and vomiting. All these toxic symptoms disappeared within two days after cessation of the medicine.

I have taken every opportunity of trying various drugs which have been recommended by colleagues, and even by irregular practitioners, but I have never found any internal treatment which gives such good results as male fern, which was, first suggested by Dr. Fouquet. At one time a Greek doctor claimed that he could cure cases by acetate of morphia, but his treatment of cases in my wards was wholly unsuccessful. Carbolic acid has been recommended, but the following case proves its uselessness: Dr. Creswell tells me he once had a bilharzia patient, who had been under treatment at the hospital three months before he tried to poison himself with carbolic acid; he became comatose, and suffered for a long time from carboluria, but the poison had no effect either on the number of eggs excreted during the coma, nor in the urine examined three months after recovery.

Until some specific is discovered we must, therefore, be content to treat the disease symptomatically.

Hæmostatic injections for hæmaturia are positively harmful, and are never used in Egypt; but early cystitis can be relieved by washing out the bladder with boracic acid or boroglyceride. Salol and urotropin (in a cupful of water) are useful for cystitis, but have no effect on the hæmaturia or on the number of eggs passed. Helmitol, which yields formaldehyde when decomposed in the bladder, has also a good antiseptic effect. Its dose is one gramme three times a-day. It renders the urine acid and is not followed by any bad effects, though care must be taken not to irritate the kidneys by any excessive doses.

Methylene blue, in a wafer containing 3 grains once or twice a week, acts simply as a bladder-analgesic, and is useful for a few of the cases which suffer from great tenesmus, and it also has the advantage of showing that the kidneys are efficient if there is no delay in the colouration of the urine. Dight (Beirut) once accidentally killed a tapeworm and several ascarides by injecting sulphuretted hydrogen into a patient's rectum, and therefore suggested that large quantities of this gas and carbon dioxide might be injected into the rectum or bladder to try and kill the worms *in situ*, for the gases are eliminated at once by the lungs without injuring the patient, but I am not aware that this experiment has ever been made.

Mr. H. Milton tells me that he has twice seen worms come away after injection of quassia infusion into the bladder; this enema might with advantage be tried for rectal cases.

Rectal cases may be treated with suppositories of belladonna or enemas of starch and opium or sulphate of copper. But tenesmus is almost entirely caused by growth near the sphincters, so that much relief can be afforded by cutting away the mucous membrane above the anus and stretching the sphincters, after which it is well to swab the gut with a solution of chloride of zinc (1 in 10), and then thoroughly wash it with saline solution. Severe cases of prolapse can be treated by lineal cautery, and if that fails, by excision in favourable cases, but it is all-important to preserve the sphincters. Growths from the vagina had better be excised.

Treatment of Complications.—It will be seen that almost all the treatment of bilharziosis is surgical rather than medical, but I may, perhaps, be allowed to give, in a few lines, a

summary of the treatment which I have seen employed by surgical colleagues.

When a calculus is present, lithotrity must be performed in spite of the existence of bad cystitis. Mr. F. Milton has lately done 124 consecutive lithotrities without a death. Lithotomy does not yield such good results—in fact, Kasr-el-Ainy hospital for many years possessed no cutting instruments—yet, in boys under four, if the stone is large, perineal or suprapubic lithotomy seems permissible. When the patient is in the last stages of the disease, with his bladder filled with sloughing necrotic growth, and the constant passage of foul urine, attended by much pain, there are only two courses, one to leave him to die in misery, and the other to drain through the perineum by Cock's puncture; but the drainage tubes may increase the sloughing in the bladder and so hasten death; a double tube should be kept in for eight or ten days, if this operation is decided on.

For acute retention of urine, when the bladder is full of clot, a perineal section must be done, because catheterisation is of no use; gently break up the clot with the finger, wash out the bladder, and the hæmorrhage will usually cease; but if not, wash again with hot or iced solution of creolin. Urethral fistulæ can be cured by free excision of the fistula and surrounding tissues. Perineal fistulæ must be scraped with a sharp spoon, and the wound left open to allow granulation; the wound should be plugged and the vessels tied, but it is not necessary to tie in a catheter. Perhaps several operations will be required, but most cases can be cured in time. A stricture, if curable, must be dilated under an anæsthetic, and then treated by the daily passage of metal sounds.

For further hints on surgical treatment, I must refer readers to Mr. F. Milton's writings, from which I have freely drawn.

Prevention.—This most important section of all cannot be written until we know for certain how the parasite gains entrance to man; but I, for one, should now hesitate to bathe or paddle in water which might be infected, and for many other reasons it is unnecessary to lay stress on the importance of drinking only boiled or filtered water. In the meantime it is obvious that eggs which have been passed in excreta should

be rendered harmless by compelling patients to evacuate them into dry earth or sand, where the embryos cannot develop, and not into water.

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ANKYLOSTOMIASIS.

Synonyms.—Uncinariasis. Egyptian chlorosis. Tropical chlorosis. Miners, brickmakers, tunnel anæmia. Hookworm disease. Ceylon anæmia. Dirt eating disease. *Fr.* Mal d'estomac, mal de cœur, cachexie africaine, Ankylostomasie, anémie des pays chauds.

The Egyptians are apt to call it, as in other countries, "mal de cœur," and almost every new patient begins the catalogue of his symptoms by putting his hand on his epigastrium and saying, "My heart." The only native name for anæmia is *Rihaqan*, or "the yellows", from an Arabic word meaning saffron.

Definition.—An insidious, wasting disease characterized by progressive anæmia without apparent cause, and by digestive and nervous deterioration, occurring chiefly in earth and brick labourers of warm climates, caused by the presence in the duodenum and jejunum of a nematode worm, occasionally proving fatal in prolonged cases, generally capable of cure upon removal of all the parasites, and capable of prevention by scrupulous cleanliness.

History.—About 3460 years ago, a medical papyrus was written in the Hieratic character, embracing in an encyclopædic form the knowledge at that time of Egyptian teachers. This came into the hands of Professor Ebers at Thebes in 1873, and has been translated into German. Joachim and Scheutbauer think that the anæmia due to the ankylostoma was well known to the physicians of those days under the names of AAA and UHA. The papyrus describes accurately, among the symptoms, heart weakness, palpitation and stabbing cardiac pain, constipation, œdema of legs, and a weight in the body pressing heavily, with other digestive troubles. But, most, wonderful of all, a remedy is prescribed for a patient *who has in his abdomen*

worms, which are produced by the AAA disease! Entozoa of many kinds are extremely common in all Egyptian patients to-day, and there is no reason for thinking they were not equally common in the days of the XVIIIth dynasty, but it is extremely improbable that any one at that time knew anything of these parasites.

Within more modern times the earliest mention of this special anæmia was from Brazil in 1648, and from the West Indies and Guiana at the beginning of the eighteenth century. In Europe the disease was first noted among the miners of Anzin in 1802, but it was not until 1838 that Dubini at Milan first discovered the *Ankylostomum duodenale.*

In 1833 Mr. Hamont, a veterinary surgeon, wrote of "cachexie aqueuse" in Egypt, which was very common among the peasants and soldiers, and which, after lasting some years, was nearly always fatal. He describes some of the post-mortem appearances, and ridicules the treatment of the French doctors, who, among other things, ordered twelve leeches to be applied to the abdominal walls of men dying from anæmia.

Pruner mentioned that he had found the parasite in Egypt; and although he added that "among adults, it is particularly the cachectic, dropsical and scrofulous who suffer from ankylostomum duodenale", he did not arrive at a correct view of the relation between the parasites and the "cachexie aqueuse" which he had himself described. Griesinger (1851) found the parasite very plentiful in Egypt, and successfully proved that it was the cause of the so-called tropical anæmia.

In 1883, at my first visit to a hospital in Cairo, I was much struck with the number of otherwise healthy recruits from the newly-formed Egyptian Army, who were under treatment for anæmia, of which no one knew the cause. Tropical diseases then had no literature, and most of us were unacquainted with the German writings on this subject, while the Egyptian professors were perfectly ignorant as to the cause of the anæmia, which we soon found was wide-spread among the gardeners and the fellaheen of the Delta. The only individual in Egypt at that time interested in helminthology was Sonsino, who told me he had written about this very parasite in 1880, and complained that he could not get the Egyptian Government to take any

interest in any scientific question. The stress of other work prevented my paying attention to this disease until 1887, when I first began treating patients at Kasr el Ainy hospital and soon after learned Bozzolo's method of killing the parasite by means of thymol. Since that time the anæmia of most male Egyptians has been recognized to be ankylostomiasis, and thousands of patients have been successfully treated in the various hospitals of the country.

In 1894 Dr. Looss visited Alexandria and Cairo in order to try and elucidate the natural history of bilharzia hæmatobia, and found, in Cairo, such a rich field of helminthological study that he was induced to return there to work at the causes of two of the most important plagues of the country, bilharziosis and ankylostomiasis. I may take this opportunity of thanking him for teaching me all that I know about the parasites which cause these two diseases.

No one who has watched his patient labours by day and night can grudge him the satisfaction of having discovered, in 1898, that the ankylostoma larvæ could enter the human body by the skin as well as by the mouth, and of having since been able to prove this fact in the most absolute way.

It was always supposed that this was the only species of hookworm which infested man, until in 1902 Stiles found, in America, a distinct species, now called *Necator americanus*.

GEOGRAPHICAL DISTRIBUTION.

Since 1878 ankylostomiasis has been found to be spread all over the north of Italy and in Sicily and Sardinia. In 1879 its discovery among the St. Gothard workmen excited the interest of the scientific world.

The disease has been found among miners in Spain, among brickmakers in Germany (Cologne and Bonn), in Belgium and the Netherlands, and at the Schemnitz mines in Hungary, where it was successfully stamped out in 1881 by enforcing cleanliness among the miners. In France it has been found at St. Etienne, Valenciennes, Commentry and Lyon.

It is worth noting that ankylostomiasis has as yet not been found above latitude 52° N., and also that above latitude 47° N.

the parasite apparently can only flourish in the sheltering warmth of mines.

It has long been known that individuals returning from infected countries to their homes might bring the parasite with them but in Northern Europe this is a danger only in the case of miners. In 1902 ankylostomiasis was found to be prevalent in a mine in Cornwall, and, on investigation, it was found that some of the miners had suffered from anæmia for nine years; it is believed that the disease was imported from one of the foreign countries from which the miners had returned, but it was impossible to settle whether the original culprit had contracted the disease in Mysore or some other part of Asia, or in Africa, America or Australia; the temperature in the mine varies from 24° to 29° C., and there were no sanitary conveniences underground. Since then the disease has been reported in a coal miner who returned from India to Glasgow.

In Asia the disease has been known since 1879 in Calcutta, and later discoveries have been made in Cochin, Lower Bengal, Assam, Burma, Ceylon, Borneo, Japan, Travancore and the Malay Archipelago. On the American Continent the disease has been found in some of the southern United States, Mexico, Central America, Venezuela, Guiana, the Antilles and in Brazil, Bolivia and Peru. "About 90 per cent of the rural population of Porto Rico are affected." It has also been found in Queensland, New Guinea, Fiji, the Sandwich Islands and West Indies. In Africa the parasite is wide-spread, and is probably present in many places from which it has not yet been reported. Besides Egypt, it is known to occur from the Senegal to the Congo, in Uganda, Lagos, Senegambia, Tunis, Algiers, Madeira, Cape Colony (Kimberley mines), Natal, Bloemfontein, Abyssinia, Zanzibar, the Comoro Islands, German East Africa, Madagascar and Mauritius.

In Egypt the disease is very widely spread, and, unlike bilharziosis, it affects the villages of both Upper and Lower Egypt. In 1893 I analyzed the first 400 patients who had passed through my wards, and found that 41 per cent came from Upper Egypt, 48 per cent from Lower Egypt, and 11 per cent from Cairo and its suburbs. In 1904 out of 261 autopsies at Kasr el Ainy, where the presence or absence of worms was

specially noted and excluding infants, this parasite was noted in 10·3 per cent; if all the deaths had occurred among country patients the ratio would have been higher. It is impossible to know what amount of the population is affected, but the returns of the recruiting commissioners for the Egyptian Army throw some light upon the number of young adult male peasants, liable for conscription, who are attacked by ankylostomiasis. Their figures show that 3·3 per cent in Upper Egypt and 6·2 per cent in Lower Egypt are rejected for obvious anæmia. Among the diseases for which conscripts are rejected, ankylostomiasis is certainly the most common. Every province furnishes rejections for this cause, Menufia being highest with 13·9 per cent, but this is only a rough clinical test, for the English recruiting officer only rejects those who are obviously too anæmic to serve in the army, and to these figures must be added several soldiers who are later invalided from the service for anæmia. The seaport towns seem to enjoy some immunity. The disease does not seem to be indigenous in the Sudan, but is common amongst the Egyptian soldiers there. It will probably spread in Upper Egypt when perennial irrigation extends further south. I have never heard of any case occurring in the English troops in Egypt.

NATURAL HISTORY.

The ensuing pages, in smaller print, are translated and abridged, by his permission, from Prof. Looss's article in Mense's "Handbuch der Tropenkrankheiten."

The nematode worm, *Ankylostomum duodenale* (Dubini), has been known by other names, *Strongylus duodenalis* (Schneider), *Dochmius duodenalis* (Leuckart), *Dochmius anchylostomum* (Molin), *Uncinaria duodenalis* (Railliet, Stiles).

It is in life a pale flesh red colour, but after death is grey or white the male is 10 and the female 12—13 m.m. long. The head end tapers slightly and contains a large mouth capsule with a horny wall, the capsule is prolonged on the mouth wall on each side of the middle line in two great hook-formed teeth, bent backward in the mouth cavity. The innermost of these bears on the inside near its base a small accessory tooth. Close by the base of the outer hooked teeth, an enormously developed single celled *head gland*, running through half the length of the worm, opens on each side; the secretion of these glands must reach the wounds made by the teeth. In the middle of the mouth capsule

there opens a gland which is buried in the œsophagus, the secretion of which apparently dissolves the mucous lining of the host's intestine. Near the floor of the mouth there are two chitinous plates, like saw teeth, which are the inner teeth rising up from the ventral wall, free into the mouth cavity. The wall of the mouth capsule consists of a number of pieces which move against each other, so that the worms can release themselves and become attached to another spot. The intestine in older worms is coloured more or less black in the anterior part, but one must be careful not to mistake this pigment for blood derived from the host's intestine.

The tail end of the body is blunt in both sexes, and in this way the female can be differentiated from the female of the *Oxyuris vermicularis*, which tapers off quite gradually to a point. The body of the male Ankylostomum ends in the "bursa copulatrix", supported by ribs like an open umbrella, which fastens the male to the female during copulation, it is, however, usually carried folded up; at the bottom of the bursa lies the ano-genital opening.

The genital opening of the female is at a distance from the anus, somewhat beyond the middle of the body. Copulating pairs, which are sometimes found at autopsies as well as in the stools after a purge show therefore the form of a Y.

The *eggs* vary between 0·056 and 0·061 m.m. in length and in thickness between 0·034 and 0·038 m.m. They are characterized by: (1) a fairly regular oval form with broad rounded poles, (2) a colourless shell which under a low power appears to be single, and is only seen to be double under a high power, (3) colourless granular contents which look grey by transmitted light. These contents are separated from the shell by a considerable space; in quite fresh fæces these contents have already divided themselves into four cells as a rule, but eggs occur with more or less divisions.

The *Necator Americanus* differs somewhat from the A. duodenale. It is thinner, its head end is bent acutely towards the back, the mouth capsule is almost round and very small with no teeth on its free margin. In place of teeth there are two sharp edged plates projecting from the side towards the middle line which are mostly covered by the thin skin of the body. The inner ventral teeth which lie at the bottom of the mouth capsule are like those of A. duodenale. On the posterior side is a single blunt cone-shaped point sticking up in the capsule cavity; this represents the opening of the œsophagus gland, and near its base on each side is a projecting chitinous plate which rises free into the mouth. The lateral portions of the bursa of the male are lengthened, so as to appear like two lappets. The vulva lies anterior to the middle of the body. The eggs resemble in appearance and size those of the Ankylostomum, but they are usually a trifle larger and they taper more at the poles.

The Ankylostomum and the Necator are both confined to human beings, for reports about the occurrence of the former in dogs and

horses have proved to be erroneous, nor does the true Ankylostomum of man occur in anthropoid apes.

Development of Ankylostomum duodenale. The eggs of the female are laid in the intestine of the host and discharged with the fæces. In the open air the embryos are developed in the eggs in a time which varies according to the temperature. The larvæ, which ought no longer to be called embryos, emerge when developed, feed on the fæces and grow. At first they have the so-called rhabditic form, i. e. the œsophagus narrows considerably in its lower half and swells again at the end into a bulb shape. Inside this bulb are three little valves which are seen opening and closing and they form a Ψ shaped figure.

Quite characteristic of the Ankylostomum larva is a long cylindrical mouth cavity which opens out like the head of a needle and is furnished with a highly refractive membrane. After some time, which again varies with the temperature, the larva, without losing its form, grows a new skin under the old one, and casts off the latter (1st ecdysis). The larva then changes its form, the body becomes noticeably thinner, the œsophagus lengthens and loses almost entirely its division into three parts and the characteristic form of the mouth disappears. The 2nd ecdysis is now beginning, the old skin rises up from the body, but is not shed for it surrounds the larva as a soft sheath, within which the animal can move backwards and forwards. The development of the larva in the open air is now at an end, and growth and feeding cease.

The *mature larva* now leaves the fæces for water or moist earth, where it may exist for months, without changing its form, living on the reserve nourishment stored in its intestinal cells; as this is gradually absorbed, the intestine which is at first granular and opaque becomes almost transparent. In still water the larva is not able to keep itself afloat by its own movements but sinks very soon to the bottom; while under favourable conditions it develops lively movements and a tendency to climb up every sort of object the surface of which is wet. Its further development takes place only after it has entered man. It is only this mature larva which is capable of developing in man, for eggs and immature larvæ, if they happen to be swallowed by man, do not lead to any infection.

Life conditions of the eggs and larvæ. The indispensable conditions for the development of the eggs are, in order of importance, *air*, *moisture* and *warmth*. In hard fæces the eggs which are on the surface develop first, those which are inside the scybala do not begin to develop until air has reached them, owing to the breaking up of the fæcal mass, otherwise they die. When the fæces are liquid from diarrhœa or have had water added to them, the eggs generally die before the escape of the larvæ. The chemical state of the fæces is probably important because an acid fermentation of the fæces will kill all eggs and larvæ. Fæces resulting from a purely vegetable diet are less favourable for development than stools following a mixed diet. In the artificial culture of eggs from fæces after a mixed diet almost every single egg can be

developed in a mixture of fæces and bone charcoal in equal parts, mixed, when necessary, with water until a stiff paste is produced; even in diarrhœa stools, good results can be obtained by adding extra charcoal.

Desiccation means absolute death to both eggs and larvæ, but the mature larvæ can live with less moisture than the eggs or the immature larvæ.

The most favourable warmth in Egypt for development lies between 30° and 35° C., but in Europe it is 25° to 30° C. (Lambinet). Lower temperatures delay development but the larvæ are not killed till freezing point is almost reached. In the summer in Egypt in an exposed laboratory temperature of 30°—35° C. the larvæ escape in 24 hours and become mature on the 5th day, while heat as high as even 45° C. does not prevent development. On the other hand, workers in Europe find that eggs and larvæ in the incubator develop badly at 37° to 40° C. In mature larvæ cold produces a distinct loss of mobility, at 15° C. they become sluggish and may lie stiff for hours, but when slightly warmed they recover their mobility.

In Egypt they can, for a short time, bear heat as high as 55° C. though they become stiff, but they regain mobility sometime after being cooled down.

Infection of man occurs in two ways by the *mouth* and *skin*. The larvæ are chiefly introduced to the *mouth* by uncleansed vegetables and by eating with dirty hands. Drinking water is less dangerous because the larvæ sink to the bottom in standing water and must first be brought to the top by stirring. On arrival in the stomach, the larvæ bore into the epithelium of the walls and so shed their sheaths, which they burst open at the head end.

On reaching the duodenum the larvæ undergo after 4—5 days a 3rd ecdysis and acquire thereby a simple spherical mouth capsule, provided at the bottom with four small teeth in the form of a cross. With the assistance of this mouth capsule they fasten on to the wall of the intestine by sucking in a plug of the epithelium as seen in Plate I.

From now onwards the epithelium cells constitute the nourishment of the worm. After another 4—5 days occurs the 4th and last ecdysis by which the worms reach their final form. They are now 3—5 m.m. long and grow rapidly, it being principally the genital organs which develop. After about 8 days these organs become mature and the first copulation follows. Copulation later is repeated several times for all the females seen by Looss in copulation contained masses of eggs and also old sperma in their genitals. After a few days the eggs are so far advanced that they can be laid, at first singly and then in numbers, so that after 4—5 weeks they are to be found in the fæces. The sexual productivity of the female, at first very great, decreases, however, in time and is always less in the older females.

When the mature larvæ reach the *skin* of a man they bore into it, leaving their sheaths behind and using principally the hair follicles as gates of entry. As the number of larvæ entering at any one time is

small this process produces no symptoms. If, however, the number runs into hundreds or thousands there is a reddening of the spot and intense burning and after some time the infected spot swells as a result of the collection of liquid in the subcutaneous tissue. The swelling, with itching, extends over a considerable area, but disappears gradually in 3—8 days.

In the hair follicles the larvæ work down to the hair bulbs and thence into the cutis. Their further progress towards the intestine, according to experiments made on puppies with the larvæ of A. duodenale and A. caninum is as follows: in the cutis the larvæ try to force themselves into the lymph vessels and later into the cutaneous veins; in these they are carried by the blood to the right heart and from there to the lung, where they pass from the vessels to the air cells and finally through the bronchial tubes, trachea, larynx and œsophagus to the intestine. This migration occurs for the most part *upon* the mucous membrane, but there may always be found in the trachea and larynx a certain number of larvæ which lie partly or entirely *in* the mucous membrane or even deep in the mucous glands. What becomes of these latter is unknown.

From the moment when they pass over to the air passages the young worms begin their further development, but the ecdysis by which they receive the temporary mouth capsule takes place apparently in the intestine only.

The larvæ which have pressed into the lymph vessels reach the blood through the thoracic duct. They must, however, for the most part first pass through the lymph glands, and there a certain number of them are rendered harmless, because the lymph cells fasten on to the bodies of the worms until they succeed in holding them fast and killing them.

After the entry of large numbers of larvæ into puppies the following results were observed: (1) œdema of subcutaneous tissue, (2) more or less blood coloured fluid in the serous cavities, (3) excessive swelling and hæmorrhagic infiltration in lymphatic glands, (4) numerous hæmorrhages about as large as a pinshead in the lungs and also in the skin near the infected spot.

In the case of skin infection the age of the animal plays an important rôle for the larvæ penetrate more easily, more quickly and in greater number to the intestine in younger animals. While quite young puppies succumb to the infection of A. caninum, older dogs show no noticeable symptoms, and the number of worms which reach their intestine remains small. In the case of man the conditions are probably similar.

Those larvæ which never reach the intestine, lymph stream or blood vessels continue apparently their wandering in the tissues of the body and may live as long as five years, or just as long as the full grown worm. If single larvæ happen to come near the skin they may cause a skin affection.

The entry of larvæ through the skin, like many another important discovery, was made by pure accident. On one occasion,

while working in his laboratory, a drop of water, containing more than 1000 lively larvæ of the ankylostomum duodenale, happened to fall on the cleft between two fingers of Dr. Looss's left hand. He was surprised to find that this was followed by redness and burning at the spot, and he wondered if this could be caused by the larvæ. He therefore allowed another drop to fall upon another part of his hand, and this was again followed by a burning sensation and redness, while examination of the drop of fluid on his hand some minutes afterwards, showed that most of the larvæ had disappeared, leaving a few sluggish ones behind, and countless empty sheaths. The observer thereupon found himself infected with the parasite. In due course eggs appeared in his fæces, and debility and anæmia followed, requiring a prolonged cure of thymol. For the last six years he has continued to handle similar cultures of active ankylostoma larvæ, but no re-infection has occurred since he has taken the precaution of protecting his hands.

The next attempt was made upon the leg of a boy, aged 13 years, one hour before the limb was amputated. The leg was thoroughly washed with soap and water, then dried; and then one drop of water containing many larvæ was dropped on the skin and left to itself without being touched. The drop spread out and dried up in ten minutes, and produced no redness of skin.

One hour after the drop came in contact with the leg the limb was amputated, and the suspected skin was at once removed from it and spread out with pins, gradually hardened in alcohol, and then embedded for section cutting. The sections when examined showed that the larvæ had entered the skin principally by the hair follicles. So far as the drop had spread there was hardly a hair follicle free from young ankylostoma.

In some there were single larvae, in others there were masses. In several cases the larvæ were seen in different stages of entry, the tail still outside, whilst the head had pushed itself between the hair and the neighbouring layer of the hair follicle.

Until they enter the hair follicle they have no purchase for their bodies, but having once entered, they progress by their own boring action. It must be remembered that the hair follicle is a recess of the corium sunk below the general surface of the

skin, and that the hair papilla is a cutaneous papilla rising up in the bottom of the follicle. The larva therefore takes a very practical way of forcing himself into the subcutaneous tissue. He never seems to penetrate into the ducts of the sebaceous glands nor into the sweat glands. In Lower Egypt, during the spring and autumn, the dorsal aspects of the hands and feet of many of the peasants are blistered by pellagrous erythema, which may help the larvæ to enter. This may possibly account for the fact that so many of the hospital patients suffer from the two diseases, pellagra and ankylostomiasis.

The next experiment was made upon a volunteer, to whom the risks of contracting the disease, and the possible complications of the malady were fully explained. As he had already been living in Cairo for some years his fæces were regularly examined for five weeks, and when it was found that no egg was present on any single occasion it was assumed that he could not possibly be already infected. Some drops of a culture containing larvæ were dropped on to his forearm, and his fæces were then daily examined, but no egg was found until the 71st day after the experiment. On several later days the well-known eggs were again seen, and are still present for he has never yet been treated by thymol.

Dr. Pieri, working in Professor Grassi's laboratory, in Italy, repeated this experiment by allowing eight drops of a culture of ankylostoma larvæ to drop upon his hand, and 71 days later he found eggs in his fæces for the first time, though Grassi and Noe, who dropped three drops of the same culture upon their hands, found no eggs on the 71st day in their fæces.

Further experiments were then made by Dr. Looss on puppies and when it was quite clear by examination on several days that their fæces contained none of the eggs of the dog's ankylostomum, their backs were gently smeared with a mixture of charcoal and fæces, in which larvæ had been hatched. On the 10th day the animals died, and at the post mortem we found great anæmia, and similar pathological appearances as after experiments on animals fed on larvæ. There were many immature ankylostomum worms in the jejunum, and no adults, showing that the infection must have been a recent one.

It thus became proved that in both men and dogs the larvæ

could enter the skin and arrive at their goal in the small intestine. But it was not until 1904 that Dr. Looss succeeded in demonstrating by what route the larvæ travelled.

Dr. Schaudinn, of the German Imperial Board of Health, considered that Dr. Looss's discoveries should be repeated independently. He therefore experimented on young monkeys, proved, by repeated examinations of their fæces, to be free from parasites; over a small patch between their shoulder blades the hair was cut close, and five or six drops of a watery suspension of ankylostoma larvæ were spread on the skin. During this time the monkey was held in position by an attendant, until all the fluid had dried, and the patch of skin had been thoroughly washed with absolute alcohol. The first experiment was made on May 28th, 1904; the animal died on June 10th, and in the upper third of the small intestine 36 living ankylostoma were found, the stage of development in all of them being from the tenth to the twelfth day. A second monkey was infected in the same way on three successive occasions, and it was killed and examined six hours after the last infection. Worms were found in the intestine, and many larvæ in the skin, some being in the hair follicles and some in the corium. Since then Lambinet has infected dogs by bringing larvæ of A. caninum into contact with the skin. Dr. Looss's observations have thus been thoroughly confirmed. Moreover Claude Smith has proved that skin infection holds good also for the Necator Americanus.

I have often been asked by students how it is that the worms can live in the small intestine without being digested by the ferments of the host. Weinland found in 1903 that the fluid pressed out of round worms from the intestine of the pig has the power of preventing the gastric and pancreatic juice of the pig from dissolving fibrin. He was able to separate some of the active substance by precipitation with alcohol, and obtained a similar body from the mucous membrane of the stomach and intestine. He therefore suggests that the resistance of parasitic worms, like the resistance of the mucous membrane of the stomach and intestines, is due to an anti-ferment which opposes itself to the tryptic or peptic ferment. This anti-ferment is destroyed by boiling.

Ground Itch (Syn. water-itch, water-pox, water-sores, sore feet of coolies). Before leaving the question of infection by the skin, I should point out that this disease is believed to be caused by ankylostoma larvæ. It is well known in Assam, Bengal, Madras, Georgia, Florida, Porto Rico and the West Indies, but has not yet been reported from Egypt, perhaps because the soil there in summer is not damp enough. It begins as a primary erythema, then a vesicular eruption, which becomes pustular, and, in severe cases, may result in obstinate ulceration or even gangrene. It is confined to the feet and ankles, it is associated with the ankylostoma larvæ in the soil, and, apparently, is caused by them. In India it only occurs during the rainy season between May and October. The natives of Assam know the disease well, and specially avoid contaminated patches of ground surrounding dwelling houses, and wear wooden sandals to protect themselves. It is found that they have less risk of this skin affection on waste jungle land, than on the cultivated ground of tea-gardens. Dr. Bentley tells me that the owner of one garden caused 60 coolies to wear boots while hoeing the garden, and none became infected. He has seen as many as 150 out of 1000 coolies affected. Further, Van Durme proved that larvæ of strongyloides intestinalis *(Anguillula intestinalis)* obtained from eggs passed in the fæces of a chimpanzee, could enter the skin of the shaved abdomen of guinea-pigs, and produce on the skin a similar eruption to that described by Bentley as "ground itch."

CAUSES.

I have shown that all that is required to produce ankylostomiasis in man is to introduce into the mouth, or rub on to the skin a microscopical quantity of earth containing living larvæ and then to patiently await the result. Unwashed vegetables, fouled by human fæces, and unwashed hands and feet coming straight from tilling contaminated soil, are probably responsible for the infection. The calls of nature are obeyed by both sexes in close proximity to the huts in the villages in, or outside which, they eat. Fortunately, continued exposure to the sun's heat during dry weather kills the larvæ, or much more harm in Egypt would be done.

But the fellaheen, who seem unconsciously to devote their lives to encouraging their entozoa, are often eaters of earth, like the anæmic negroes of the West Indies and Guiana; 26 per cent of my cases confessed to eating it, though most of them allowed that it was a bad habit and induced anæmia. They call the Nile mud "teen Ibliz" (πήλος, mud), and apparently have some old superstition that the soil, so fertile to vegetation, cannot but be beneficial to the human economy. Chlorotic and pregnant women often eat earth, as also the women who clean the corn after threshing. There is, too, a curious custom connected with earth eating, for on the day of maximum high Nile, and the general rejoicings thereupon, the town crier, who is on the look-out for backsheesh presents *teen ibliz* with a lemon to the inhabitants for luck, and many of them eat it.

When once they become anæmic, they also eat other strange things, including unripe maize, and I have sometimes wondered whether this is one of the reasons why so many of the fellaheen suffer from both pellagra and ankylostomiasis.

Age.—Nearly all the patients, or 77 per cent of them, are in the prime of life, between 20 and 40 years of age, at the very time when they ought to be of the highest value to the State and to their families. The youngest case I remember seeing was a child of four years; the disease in boys of about twelve is common, but above 60 years of age it again becomes rare.

Sex.—Out of 1000 cases only one per cent belonged to the female sex, such cases being almost invariably girls just under the age of puberty, or fellah women who work in the fields with their families. Many more peasant women suffer from the disease than these figures suggest, but they have not yet learned to apply for hospital relief in the same proportion as the men. Many women in Cairo suffer from anæmia, but this is dependent upon their lack of exposure to fresh air, confinement to insanitary houses and to the habit of wearing a veil over their faces when out of doors, to unsuitable food and, in the case of some mothers, to prolonged suckling; in the fæces of such town dwellers I have often searched for the eggs of the parasite without finding them. In Porto Rico out of 5,490 patients 40·6 per cent were females.

Nationality.—The bulk of the patients are Egyptians, and I have not yet met the disease in a Bedawi living in the desert, though several of the Bedawin living in villages near the Pyramids have come to me with this disease. There are comparatively few cases among the Berberins and Sudanese, and the experience of the Egyptian Army is that anæmia exists only to a small extent among the black troops, and, when it occurs, it is specially among those recruited in the Delta. It must not be forgotten that both the Berberins and Sudanese met with in Egypt are distinctly cleaner in their habits than the Egyptians, and also that they do not naturally lend themselves to agricultural pursuits like the natives of Egypt. (The negroes in Porto Rico enjoy some immunity). I have seen a very few cases among Greeks of the poorest class.

Occupation.—The hospital patients spring from the poorest classes of the community, but there is, of course, no reason why anyone should not contract the disease if he subjects his mouth or his skin to the contact of foul earth containing living larvæ. I have seen several cases among medical students, dating from the time when they lived in the country with farmer relatives, and from distant parts of Egypt a few private patients have come to see me begging to know whether their symptoms were caused by ankylostomiasis; whenever I have found any of these infected they have been men who have worked on the land with their own hands. Among 200 men of whom I have careful notes no fewer than 190 were accustomed to work in more or less damp earth with their hands, and sometimes with their feet.

The actual figures are as follows:—Fellaheen, working all day in the field with their hands and with the hoe, 152. Masons or bricklayers' labourers, building walls with mud bricks and mud cement, 18. Scavengers of street refuse and of cesspools, accustomed to emptying with their hands the dry contents of the latter, 7. Pedlars who dig up and afterwards sell unwashed vegetables, such as onions and radishes, 7. Limestone carters accustomed to act as scavengers on their return journeys to the desert hills, 3. A gardener, a fisherman in mud, and a "shaduf" worker on the Nile bank, completed the 190 cases. The remaining 10 men were 1 coffee-stall keeper, 3 readers of the

Koran, 1 blacksmith, 1 street shoeblack, and 4 street beggars, 2 of whom announced that they were habitual earth eaters. All these denied ever having done a day's work in the fields, and, excepting the Koran readers, were all townsfolk. These readers, like the beggars, live on food which is given them by the well-to-do. Outside the large towns nearly every representative of the lower classes works in the fields, either for his own profit or for hire. Many policemen, incapacitated temporarily by this disease, have passed through my hands, all of whom dated their illness from periods of their life when working as fellaheen. Some of these had joined the police direct from their villages; others had previously passed through the army.

The Egyptian peasant works all the day with his naked feet, legs, and hands exposed to a coating of damp mud, containing ankylostoma larvæ which have sprung from the eggs contained in the fæces of infected natives. Latrines are unknown in the country villages and every man eases himself whenever and wherever he thinks fit. The entrance of larvæ into the human skin affects many countries besides Egypt, and may account for some of the infection reported from India, the Italian rice fields, St. Gothard tunnellers, and mine labourers in Europe where men work with bare feet in infected mud and water.

Season.—I have already said that the larvæ require a moderately high temperature for their development, which accounts for the confinement of the disease to warm countries, and to the fact that in Northern Europe it can only prevail in the mines, where the temperature is higher than that of the outer air. There are no statistics to show whether certain months are more dangerous than others to the fellaheen, but we may assume that the damp, hot months are the most important, because the larvæ are so much more active then.

Actual damage to man caused by worm.—It used to be believed that the worm was purely a blood-sucker, and that the anæmia was caused by loss of blood. But Looss finds that the mucous membrane of man's small intestines is the only food of the worm. He points out that the plug of mucous membrane is not only sucked into the worm's mouth, but actually disappears, and can be traced down its œsophagus,

PLATE I

Longitudinal section through head and neck of Ankylostomum attached to intestine of host. Some villi have already been eaten by the worm. A plug of submucous connective tissue has been drawn into the worm's mouth cavity and continues into the œsophagus. The lines of traction in and around this plug are all towards the mouth. 1 × 110.

Photographed by Dr. Bitter 6 hours after death of the host. (*Mense's Handbuch.*)

and sometimes even to its intestine; moreover, in most cases the entire contents of the worm's intestine are composed of the histological elements of the host's submucosa, and when these are mixed with blood, the complete digestion of the blood corpuscles does not occur, for they retain their colour and only the plasma becomes more or less indistinct. While the worms are eating into the mucous membrane, if they accidentally meet with a blood vessel, its wall is torn open, and the blood which escapes is partly swallowed by the worm and partly flows out beside it.

Looss further suggests that man's anæmia is chiefly caused by some toxin introduced from the large and important head glands of the worm.

SYMPTOMS AND COURSE.

Ankylostomiasis is the name applied to the group of symptoms caused by the presence in the small intestine of the parasite under consideration, and the term must be employed in a general way as indicating a condition in which the symptoms of the disease can only be accounted for by the proved presence of worms in the body, or eggs in the fæces. The worms are sometimes able to inhabit men for months, or even years, without causing severe anæmia, and in India, as well as in Egypt, persons have been known to harbour as many as 100 of these worms without showing any marked symptoms of illness. The mere presence of a few ankylostoma in the intestine cannot be said to constitute a typical condition of ankylostomiasis, but, sooner or later, the well-known symptoms will begin to manifest themselves.

These may be grouped into three classes,—those affecting the digestive, circulatory and nervous systems. They usually affect the patient in the above order, but most hospital cases are so far advanced before they come under treatment that all three systems are affected. The worms are seen so seldom in the fæces that patients never know that they are suffering from entozoa, unless they happen also to be the hosts of Ascaris, Oxyuris, or Tænia.

Gnawing, throbbing pain in the epigastrium is the first symp-

tom complained of, chiefly because it is constant, whereas severe colic and borborygmi of intestines are only present from time to time. The appetite is invariably affected, sometimes ravenous at the beginning, but later always capricious and diminished. I have already referred to earth hunger, sometimes the cause, and sometimes the effect of the illness.

Vomiting and nausea are rarely complained of. *Constipation*, when not under thymol and purgative treatment, is a very constant symptom in hospital. Sixty per cent I found had suffered for a long time from obstinate constipation, 28 per cent had had diarrhœa before admission, and 12 per cent had no recollection of being troubled with either. None of the figures depending upon the memory of the patients must be taken as absolute truth, as the intelligence of many is of a very low order. Diarrhœa and even dysentery are not uncommon in very advanced cases, especially those complicated by Bilharziosis of rectum, and unless the patient is robust enough to support thymol such cases are apt to end fatally. However, I have had the satisfaction of curing some cases of ankylostomiasis which had been unsuccessfully treated elsewhere for several months as chronic dysentery. *Flatulence* of the lower abdomen is present to a slight extent in one third of the cases. The *urine* is not unlike that of ordinary anæmia, neutral or alkaline in equal proportion, and rarely acid, pale coloured, with specific gravity ranging from 1010 to 1015. A trace of albumen is present in all the most advanced cases, without casts under the microscope. Little is gained by naked eye examination of the *fæces*. Blood is seldom seen, at Porto Rico out of 22,000 specimens of fæces examined by the Commission, only 6 contained blood.

Pain in the head, generally referred to the temples, while in the knees there is invariably present great weakness and some pain; occasionally there is in addition pain in the shoulders. *Giddiness* is another very general symptom, and it is this as much as anything else which compels them to give up work. *Palpitation* over the heart, in the epigastrium, and in the temporal arteries is sure to be present in bad cases, while the anæmic murmurs of heart and neck are solely dependent upon the degree of anæmia, and can be banished by a prolonged

course of iron. Hypertrophy of heart was noted and verified after death in some of the advanced cases. *Dyspnœa* and noises in the ears are present, as might be expected with marked anæmia. *Œdema* of face and legs, like albuminuria, is only to be seen in the worst cases, and some of these have general œdema and pericardial effusion when admitted in a dying condition. The tongue is extremely pale, the mucous membranes excessively white, so that it is almost difficult to see where the mucous membrane of the lip ends and the skin begins, the face has an unhealthy yellow pallor, very different from the ruddy brown of the peasant in health, and the bloodless nails testify to the blood containing not more than half the normal number of red corpuscles.

The skin is always cold, and the temperature before thymol generally subnormal in uncomplicated cases. After excluding any fever produced by concurrent diseases, and any defervescence caused by thymol, I find that one third of my patients had a normal temperature during their stay in hospital, but that two thirds had a distinctly subnormal range, varying from an average of 36°·3 C., a.m., to 36°·9 C., p.m.

Many of these patients when convalescent had an increase of half a degree, night and morning.

After eliminating all those who had fever while in hospital, (so as to exclude the possibility of malaria), or a history of intermittent fever or any enlargement of spleen, I found that 68 per cent of the remainder stated that their troubles had begun with a few days' fever.

The *eyes* of patients show a pearly white conjunctiva, singularly in contrast with the yellow colour of the face.

The refraction tests of the eyes show astigmatism in many cases, but probably not in greater numbers than among the other fellaheen. There are no ophthalmoscopic lesions, except that in about half the cases the fundus is very pallid. I have never found any retinal hæmorrhages, epistaxis, nor general tendency to loss of blood, but, in some very rare cases, there has been melæna while under treatment in hospital. Jaundice is not one of the symptoms.

In 1902 it was suggested by Dr. Delamere of British Guiana and others that ankylostomiasis could be diagnosed by a pecu-

liar marking on the tongue, "exactly as if the patient had just wiped a penfull of Stephen's blue-black ink on his tongue," he further stated that the patches gradually fade away from the tongue, after appropriate treatment with thymol and iron. I was under the impression that the brown or black patches of pigment on the tongues of some coloured patients were no more frequent in ankylostomiasis patients than in other hospital cases. However, I examined 100 in-patients in whose fæces ankylostoma eggs had been found, but only 11 had any patches of pigment on the tongue, though 53, or more than half of them, had distinct colouring of pigment in the sclerotic of their eyes. I then examined 100 other patients in the wards in whom no trace of ankylostomiasis could be discovered: 7 of these had pigmented patches on the tongue, and 27 of them had evident pigment in the sclerotic. This eye pigment varied from "very little" to "very marked," and is sufficient to show that I was really examining dark-skinned patients. As a matter of fact, the individuals examined varied from the *café-au-lait* coloured Egyptian to every shade of brown Nubian and black Sudanese. I took the cases who happened to be in hospital without exercising any choice; they were all adults, and very nearly all were men.

I am now prepared to state that in Egypt, at least, the marks on the tongue have no reference to the stage of anæmia, and the observation is of no value for diagnostic purposes.

Sleepiness is decidedly a symptom, and among the worst cases are those which are always asleep, and which cannot be interested in anything when they are awake. The *facies* of the patient is characteristic, and there is a dense stupidity, associated sometimes with reiterated demands for a favour already granted, but so many of the Kasr-el-Ainy cases are complicated by pellagra that it is difficult to try and get a pure picture of the mental state of ankylostomiasis patients.

Debility and wasting are, of course, important symptoms, but it must be remembered that when a man is so weakened by the disease that he cannot work he ceases to earn money, and his food supply becomes limited. In fact, it is very often poverty which brings the patient to the hospital. The average weight of 100 grown men upon admission to the hospital was

117·5 lbs. The average height was 5 feet 5½ inches, which, by Dawson's tables, should scale at least 135 lbs. Of the patients who stayed in hospital more than two weeks 70 per cent gained weight, 22 per cent lost, and 8 per cent remained stationary. The average loss of weight was 3·2 lbs., and was, of course, caused by the necessary starvation, thymol, and purging. The average gain of weight was 5·4 lbs., some gaining as much as 15, 17, 18, and 20 lbs.

Impotence seems to be a decided symptom of ankylostomiasis. I found that 63 per cent of the adult patients were completely impotent, and 13 per cent were partially so, also, the disease delays the appearance of puberty in both sexes. Advanced cases, even when they show no sign of concomitant pellagra, have abolition or diminution of the knee jerk.

The *skin* is not often affected in Egypt, but I have seen a few cases of urticaria, apparently due to recent infection, and similar to the "bunches" which have recently been found on the buttocks, knees and forearms of infected miners in Cornwall.

The only other skin affections are a tendency to small boils and general pruritus, specially marked over the trunk, but both these are among the rare symptoms.

Examination of the blood.—A detailed blood examination of several cases, such as that made by Boycott and Haldane confirms one in the belief that ankylostomiasis anæmia is not directly due to loss of blood from the punctures of the worms. As a matter of fact, the patients really suffer from a condition of hydræmic plethora. This special anæmia differs greatly from that due to hæmorrhage, or that of pernicious anæmia, but it closely resembles the anæmia of chlorosis, the chief points being an increase in the total volume of blood, a varying increase in the leucocytes and some increase, relative and absolute, in the eosinophiles.

I have notes of 173 cases in which the blood-corpuscles were counted on admission to hospital. In only three patients were the red corpuscles found to exceed 4,000,000, and even then the excess was extremely slight. Twenty-three per cent numbered between 3,000,000 and 4,000,000 to the cubic millimetre, 46·8 per cent varied from 2,000,000 to 3,000,000, and 28·3 per cent had less than 2,000,000 blood-corpuscles. The

minimum recorded was 930,000 in a patient who afterwards did well. The average gain of red corpuscles during stay in hospital of these cases was 1,290,000, and could have been much more if it had been possible to keep the patients for a longer time.

The least gain recorded in any one patient was 310,000, while ten patients gained over 2,000,000 during their stay. The youths seem to have the most recuperative power in this direction, and I recorded an increase of 2,542,000 in a boy of eighteen years. Another boy, aged sixteen, during one month gained seven pounds in weight and 2,208,000 red corpuscles; while an Alexandrian blacksmith, in six weeks, gained eleven pounds in weight, and raised the number of his red corpuscles from 1,054,000 to 3,875,000.

The average number of *white corpuscles* upon admission was 10,360, and the average increase in hospital was 5,370.

One of the interesting points is that during the process of cure the number of leucocytes per cub. mm. increases, for this goes to disprove the suggestion that there is no real leucocytosis in ankylostomiasis, or that the increase of leucocytes is due to intercurrent diseases. It will generally be found that the highest leucocyte counts are in patients with a short history of illness, and probably recently infected, while the old standing cases exhibit low counts of leucocytes.

In the differential count of leucocytes the great feature is the absolute and relative increase in the number of eosinophiles which were found, in Cornwall, to vary from 11 to 72, while the absolute number in 16 cases varied from 676 to 32,000 and there was sometimes some increase in the mast-cells.

In my cases Gowers' *hæmoglobinometer* showed an average of 26 per cent upon admission, the numbers varying from 10 to 54 per cent of hæmoglobin. The increase of hæmoglobin in hospital ranged from 22 to 32 per cent, and probably continued when the men became out-patients.

As a further blood test, Dr. Kaufmann examined twenty typical cases for me to determine the volume of the red corpuscles. Healthy men were found to have from 45 to 50 per cent by volume, but the anæmic patients had only from 15 to 43 per cent., the average of all being 25 per cent. This

average corresponds very fairly with the average of red corpuscles by enumeration.

One patient's hæmoglobin was raised from 35 to 67 per cent in two weeks, and his volume of red corpuscles from 31 to 45 per cent.

Men can perform hard manual labour in spite of their hæmoglobin being reduced to under 40 per cent., and some poor wretches have tried to work when it was as low as 20 per cent.

The following summary shows the figures in a tabular form:

Count	*Normal*	*Ankylostomiasis*
Red cells	4½—5 millions	1—3 millions
Hæmoglobin	90—100 %	10—54 %
Colour Index	·9—1	·5
White cells	6,000—10,000	9,000—12,000
Eosinophiles	2—4 %	23 %
Eosinophiles absolute number	225	3,000

Average stay in hospital.—This must depend upon how far the wards are crowded and also upon the willingness of men to remain under treatment during convalescence, for most of the fellaheen have families dependent on them, and are obliged to return to their villages to support them. Some of my patients spent in hospital periods varying from 14 to 76 days, the average of the whole being 30 days. Upon leaving the hospital they were cured of their parasites, but not entirely of their anæmia. Some were discharged in order to make room for other inmates, but most of them felt so much better in health that they clamoured to return to their work.

Duration of illness.—Nearly every patient said he had been ill two or three years before admission. I find among my notes 29 cases, uncomplicated by other diseases, where the patients said they had suffered more than three years. Of these, 13 had been ill for four years, 6 for five years, 3 for six years, 2 for eight years, 2 for nine years, 2 for eleven years, and one vowed that it was fifteen years since the commencement of his symptoms.

It may safely be said that during these long intervals of time very few patients had any rational treatment.

COMPLICATIONS AND CONCURRENT DISEASES.

It is very rare to perform an autopsy upon an Egyptian fellah without finding various entozoa, and the ankylostoma patients are no exception to this rule. *Bilharziosis* is, of course, the most common disease, and in experimenting on cases of bilharzia with thymol I have often found ankylostoma, though the patients had no symptoms of anæmia.

Oxyuris vermicularis is present in about half the patients and is usually expelled by the first dose of thymol. *Ascaris lumbricoides* is the next most common, and it also is expelled by thymol. *Taenia mediocanellata* is only present in 1 per cent of the cases, and is not well acted on by thymol. *Tricocephalus dispar* is even more rare as an accompaniment.

Favus of the scalp is very often present, and I have already referred to the common presence of *pellagra*.

Among concurrent diseases often met with are: intermittent fever, dysentery, fistula in ano, hæmorrhoids, vesical calculus, chronic bronchitis and pulmonary tuberculosis.

Albuminuria occurs in 8 per cent of the advanced cases, besides those in which there is kidney disease due to some other cause.

Ankylostomiasis often leads to a fatal result in women after parturition, even in the absence of any unusual hæmorrhage.

MORTALITY.

Of my hospital cases 89·5 per cent were cured, or greatly relieved, 2·5 per cent were unrelieved, and 8 per cent died. Of the deaths, about one third were admitted to the hospital moribund and died within a few hours, having generally been found by the police lying in the streets starved with cold and hunger and in the last degree of exhaustion. Another third die from other diseases, such as enteritis, dysentery, kidney disease and general tuberculosis, and the remaining third die from ankylostomiasis, complicated by pellagra and bilharziosis. Some of the richest helminthological finds in the post mortem room may come from surgical wards, such as a man who had 13 bilharzia worms in his portal vein, while his intestines con-

tained 3 ascarides, 3 oxyurides and 118 ankylostoma, 50 of which were still attached to the jejunum. Most fatal cases of ankylostomiasis have, before death loud anæmic murmurs, markedly subnormal temperature, slight general œdema, albuminuria and great mental weakness. The actual cause of death is exhaustion from cardiac degeneration and anæmia.

PATHOLOGY AND MORBID ANATOMY

The skin is lemon coloured. There is usually some absence of subcutaneous fat, the muscles are always very pale. Œdema may be present in the lower extremities, and more rarely may be general. Serous fluid in great excess is found in the pleuræ, pericardium, peritoneal cavity and ventricles of the brain. All the organs are extremely pale and bloodless on section. The lungs are œdematous. The *heart* is sometimes hypertrophied, sometimes very small, but in advanced cases shows marked fatty changes and is speckled like the breast of a thrush, and in about 12 per cent there are marked changes in the mitral valve. There are no characteristic naked eye changes in the *liver*, the organ is not enlarged, is very pale, and of a light brownish yellow colour. The spleen is often slightly enlarged, even when there is no history of previous malaria. The *kidneys* show such typical change that we have got into the habit of referring to the "ankylostoma kidney." They are large and pale, the capsule easily separable and the surface pale, mottled and anæmic. At first sight the section looks like chronic parenchymatous nephritis, but on closer examination you see that the fatty degeneration is universally distributed and not confined to the cortex. The microscope shows an advanced general fatty degeneration, not specially limited to the convoluted tubules. A few of the patients, instead of the large, pale kidney, are found to have granular kidneys with several small cysts. The brain is always exceptionally white.

The small intestines show, of course, the most important changes. As a rule there are many hæmorrhages and bites in the jejunum and ileum, but in one case, where the bites were carefully counted, there were only six visible to the eye in the jejunum and ileum, and no worms were found. In another

case, however, there were 575 bites in the small intestine, besides 250 ankylostoma. In yet another there were 100 bites the farthest of which was 4½ metres from the pylorus. In very few cases is there liquid blood in the intestine.

I have not observed the pigment in the stomach, small intestines or mesenteric glands found by some writers, but it sometimes occurs in the rectum and lower colon as a legacy of former dysentery.

The great variability in the number of ankylostoma found at the autopsies is interesting. In some cases, treated by thymol, no worms can be found, in other cases, where thymol has not been used or insufficiently used, there are worms varying from 10 to 50, and sometimes we find as many as 200 or more.

For instance, in one autopsy I counted 381 ankylostoma, and in another 863. On that occasion the worms were scattered from a point 3 cm. beyond the pylorus for the length of three metres; 217 of the 863 were still attached to the intestine, and were surrounded by much bloody mucus, while 646 were lying free, 16 of the latter were still alive, and one couple were in copulation.

The maximum record was reached in Case 1., where 1524 worms were counted.

The position of the worms must be noticed, for it is quite the exception to find any parasites in the duodenum. I think this can be explained by remembering that the duodenum becomes riddled by the ravages of the earliest generations of the worm and the villi must, in time, get eaten away. It would, therefore, be quite natural for succeeding generations to shift their pasture from the duodenum to the jejunum, and this may partly explain the interesting fact that it is not the most advanced cases of anæmia which always yield the largest quantity of worms. Or is it that the half-starved worms are dislodged by purges or repeated attacks of diarrhœa? The furthest feeding-ground that I have ever seen was 6·30 metres from the pylorus, where there was a worm firmly attached. But, as a rule, the attached worms are all within 2 metres of the pylorus, and have their heads and sometimes half their bodies buried in the mucous membrane. It is often impossible to dislodge them by a strong stream of water, and they must then be pulled out by forceps.

I once examined about fifty cases to see the proportion of male to female worms, and found it 56 to 44 per cent.

The bone marrow of the femur has only occasionally been examined, but when seen it has usually been paler than normal, of the colour of strawberries and cream.

The following case is instructive as showing how a man may become reinfected, and even die, if he returns to his former habits after an apparent cure.

Case 1.—A. G. aged 35, an Egyptian carter of fellah origin, was admitted as an ankylostoma patient under my care in 1900. After the routine treatment with thymol and sulphate of iron, he left the hospital convalescent, and remained fairly well for two and a half years. About the beginning of 1903, however, he noticed that he began to suffer from headaches, which he recognized as being similar to those he had before, and very soon afterwards he complained of palpitation, giddiness and great fatigue after the slightest exertion. These symptoms were so marked that the patient himself was quite aware that he had a return of his former trouble, but, for some reason or other, he delayed coming to the hospital until he was forced to do so by excessive weakness. He was readmitted in May 1903, and placed in one of Dr. Tribe's beds. He presented every appearance of great anæmia and prostration, and had hardly the strength to turn round in bed. He complained chiefly of great weakness, feverishness, headache, palpitation, anorexia, vomiting, pain in the epigastrium, constant giddiness, thirst and drowsiness, with a general feeling of misery and depression. He had some diarrhœa, and the motions were found to contain a large number of ankylostoma eggs. There were pain and tenderness over the hepatic and splenic regions. He was too ill to be examined very thoroughly, far too weak for the thymol treatment, and he died a few hours after admission. These post mortem notes are condensed from Dr. Symmers's report: Body very emaciated, heart contained a quantity of very pale blood, which clotted after lying on the table for a short while; the muscle of the left ventricle was in an advanced condition of fatty degeneration; that of the right ventricle was very anæmic and pale, evidently fatty, but did not show speckling. The aorta showed very faint, atheromatous patches,

a few of which were also present on the mitral valve, the coronary arteries were normal. The *lungs* were very anæmic, the bronchial tubes being very blanched. The *liver* weighed only 1150 gms., was small, tough, and of a very pale fawn or light brown colour, and, on section, had a somewhat speckled appearance. The gall bladder was half full of thick, dark brown bile, and its wall was stained greenish. No ascites nor œdema. The right *kidney* weighed 150 gms., and the left 130 gms., both were extremely anæmic and somewhat tough, the capsule came away fairly easily; on section the cortex of each was fairly normal, or perhaps slightly reduced in size, and the interior of the organs was extremely pale, but not mottled. The spleen, 125 gms., was pale, and the trabeculæ were very manifest. The intestines contained a great number of ankylostoma, which were numerous in the duodenum and for two metres down the gut, from thence onwards the small bowel contained a large amount of blood clot and semi-clotted blood, but very few worms. The large bowel contained a quantity of blood clot, in which a few worms were embedded. No bilharzia worms were found. The *brain* was extremely anæmic, and weighed 1030 gms. Worms were so numerous that it was considered worth while to count them; besides many which must have escaped, 1524 were counted, and of these, 1353 were present in the first two metres of gut after the pylorus.

I have said enough to show our belief that the worm introduces some toxin into man while biting him, and it is probably this toxin which develops in the human being and causes his illness and, sometimes, his death. It appears also that the toxins in the urine of ankylostomiasis patients can conduce to anæmia, for Lussana, in 1890, condensed urine at 60°—70° C. to the thickness of syrup, extracted the residue with pure alcohol and dissolved the extracted water after evaporating the alcohol. He then subcutaneously injected rabbits on eight consecutive days with this fluid, and induced in them loss of colour of the red blood corpuscles, increase of fibrin and poikilocytosis. When the injections were discontinued all these symptoms gradually disappeared. He found that urine, passed by patients not suffering from ankylostomiasis, but similarly treated, did not produce such symptoms.

DIAGNOSIS.

The anæmia is very well known to the patients themselves, but as the worms are not, under ordinary circumstances, to be found in the fæces, they do not know the parasitic cause of the anæmia, and until about 1893 most doctors in Egypt were unaware of the intimate connection between the worm and the symptoms. I have, therefore, often ventured to insist that cases should be returned in hospitals as ankylostomiasis, and not merely as anæmia. Even after thymol and a purge have been given to a fasting patient, care and patience are required to find the *worms*. This can easily be done after submitting the fæces to several washings with clean water, and examining the final deposit. The worms are generally discoloured by the fæces, and must be carefully distinguished from any accompanying oxyurides. It will be found that thymol has removed most of the fæcal odour.

The microscope furnishes the readiest means of discovering the parasite, for in most cases *eggs* can be found in a tiny portion of the suspected fæces, which, to make the examination more agreeable, may be washed in a test-tube with a weak solution of carbolic acid.

"*Strongylus subtilis* eggs may be mistaken in fresh fæces, but they are larger, and their contents consist of 20—30 small cells. *Oxyuris vermicularis* eggs may be excluded, because of their small size, their thick and double shell, and the absence of the free space between the shell and the cells. The *Ascaris lumbricoides* egg is known by the crenated yellow coating of the outer shell, while the inner shell is thick and double, and at the time of deposit contains a single cell which only begins to divide after some days. Free moving larvæ in fresh fæces are never Ankylostoma but are probably *Strongyloides stercoralis*. When the fæces are not quite fresh, and the temperature high, Ankylostomum eggs may be found in an advanced state of development, and after 24 hours living larvæ may be present, but even then the ordinary eggs can be found inside a lump of solid fæces. The immature larvæ can be recognized by the characteristic form of their mouth cavity. The mature larvæ lack this, but can hardly be confused with

ankylostoma were passed. On May 26th 18 more appeared after a similar dose; on June 2nd there were 3 more; and on June 8th a last detachment of 5. Two more doses of thymol were given without result, and eggs ceased to be found under the microscope. In order to see whether thymol had cleared out all the round-worms, a dose of santonine was given on June 5th without any effect.

Another successful result was the following; *Case 2.*—A. M. aged 33, was admitted from the out-patient department on April 29, 1897. He was a fellah from Assiut, but had been working as a mason in Cairo for the last two years. He was extremely anæmic and said he had been so for about twelve years. His weight was 138 lbs. He confessed to pellagrous symptoms for the last twelve months, and must have had them decidedly longer, for there was slight roughness of skin on the back of his hands, forearms, elbows, and on his feet, legs and knees; he could only sleep four hours at night, had forgotten how to smile and was very sad and melancholy, while his knee-jerks were much too brisk, and he had tenderness on pressure on both sides of his fifth dorsal vertebra, his tongue was very pale and denuded of epithelium. On May 2, after his first dose of thymol 523 ankylostoma, and 55 oxyurides were found in the motion by Dr. Looss. On May 7th, after a second dose, there were 3 oxyurides, but no ankylostoma, and after a third dose, given because I thought the result too good to be true, no worms were passed, and no eggs could be discovered. This individual was, therefore, fortunate enough to be rid of all his important parasites in one day. Nothing, therefore, remained for him to do but to take tonics and get well of his pellagra.

In 229 patients I have induced students to count the numbers of ankylostoma voided after the first dose of thymol. There were 14,190 of this species alone, giving an average of 62 per man. The largest numbers met with were 350, 372, 480, 483, and 600. On seven occasions no worms at all were found, in spite of eggs under the microscope, but in three or four of these patients a second dose of thymol produced a very few specimens.

Now one word about the number of doses of thymol required

to completely eradicate the parasite. It is true that the bulk of them are brought away by the first dose, but there are invariably many stragglers behind, and it seems dangerous not to attack them. In 184 patients I was able to prove the absence of worms by giving at least two and sometimes more doses of thymol after the last appearance of the parasite in the stools. The average number of doses required proves to be 2·6. Forty-two men were cured after only one dose, 58 after two, 42 after three, 25 after four, 9 after five, and 4 after six doses of thymol. In two cases, however, the parasite could only be entirely dislodged by seven attacks, and two other cases required eight doses of thymol. In the event of patients being treated outside a hospital, it will, therefore, be advisable to give the thymol treatment on at least three occasions. The dose of thymol is of considerable importance. I quite agree with other writers that small doses are valueless. I used at first to give 6 grammes at intervals of 2 grammes every two hours, but to prevent unnecessary collapse I soon reduced this to 4 grammes as an adult dose, viz. 2 grammes at 8 a.m., and the remainder at 10 a.m. I found that 4 grammes were as efficacious as six, and are certainly less dangerous. But even this reduced dose produced, in some patients, alarming collapse, and we then tried to find the minimum dose of thymol which could expel the parasites. The routine treatment at Kasr-el-Ainy for some years now has been to place the patient on fever diet for the whole of one day, to give him one gramme of thymol in a cachet at 6 p.m., when he is already in bed, to give him a second gramme at 6 the following morning, and to follow this at 8 a.m. by a dose of 30 gms. of sulphate of magnesia; he is then able to take his ordinary dinner at 12 o'clock, and the motions are preserved to see how many worms have been passed. We also made it a rule that the patients should lie down flat in bed after their first dose of thymol, and not move out of bed on the second day without permission. Previously to the enforcement of this rule, patients used to be occasionally brought back to the wards from the lavatories in a semi-fainting condition. The sulphate of magnesia after the thymol brings away any dead worms, and, what is more important, prevents the thymol from remaining too long in the

digestive canal. In my first trials I used to give a purge before, as well as after the thymol, but I soon omitted the preliminary aperient as being unnecessary, and as contributing not a little to the patient's exhaustion. I soon substituted magnesia for castor oil. Alcohol, ether, chloroform, glycerine, turpentine and oils are all solvents of thymol, and therefore must not be allowed to enter the alimentary canal while the thymol is there. Nurses must therefore be warned not to give brandy to a collapsed patient, but strychnia hypodermically, coffee by the mouth or hot water by the rectum may all be given, when necessary, with advantage. Thymol is probably very insoluble in the stomach and duodenum, but affects some patients much earlier than others. No patient should be given the second part of the thymol dose until it is certain that he is not in any way unpleasantly affected by the first part. Occasionally patients vomit after swallowing the thymol, but as a rule they retain it perfectly, and have great faith in its properties, for they often ask for an extra dose of it while convalescing.

Large doses of thymol have poisonous effects upon the system, not unlike those produced by carbolic acid. The temperature is lowered 1° or even 2° C., and both pulse and respiration are slowed. The patient remains for a few hours collapsed, giddy, and faint, and has to be kept lying down, but in a few hours he is quite well again and asking for food. The following case is a typical one in a very anæmic man.

Case 3.—January 14th, 1892—6 a.m., temp. 37·5°, pulse 80, resp. 19; patient in his usual state, and was given two grammes of thymol. 7 a.m., temp. 37°, pulse 80, resp. 19, says he has slight nausea, giddiness, and colicky pain at the epigastrium. 8 a.m., two grammes more of thymol given. 9 a.m., temp. 35·5°, pulse 70, resp. 17; great giddiness, cannot stand or walk. Very sleepy, and talks like a drunken or very sleepy man. 12 noon, symptoms much the same; sweating while asleep. 2 p.m., temp. 37·5°, pulse 75, resp; 18; apparently quite well again. Says he does not mind the thymol, except that it makes him lose consciousness.

The following case is worth quoting very briefly, because of the low temperature, and of the disease not having been previously recognised.

Case 4.—December 29th, 1891.—A fellah was admitted from the Barrage, near Cairo, complaining of giddiness, pain in his head and epigastrium, and weakness of knees. He was extremely anæmic and his face very yellow; but his nails, though very white, showed a faint pink tinge. He seemed only half-witted, but I learnt that he had been ill for three years, and had been treated by rustic quacks for hæmorrhoids, and had also been treated four times in a Government hospital for dysentery. His motions, which were five in the twenty-four hours, contained many ankylostoma eggs. He had a loud anæmic murmur; liver and spleen slightly enlarged; urine 1013, no albumen.

Dec. 30th.—7 a.m., one hour after thymol, temp. 36·6°, pulse 62, resp. 20. 9 a.m. after 2 more gms. of thymol, temp. 35·4°, pulse 58, resp. 16; pupils contracted, great weakness, sleepiness, giddiness. 11 a.m., having now taken six grammes of thymol, temp. 35° (95° F.), pulse 46, resp. 16, collapsed, and coffee and strychnia had to be freely administered. 2 p.m., quite well again, temp. 36·6°. This man was discharged on May 31st, 1892, with good colour, looking happy, though there was still slight mental weakness. His diarrhœa had completely ceased, his weight had improved from 84 lbs. to 101 lbs., and his temperature was no longer subnormal.

The contra-indications for thymol are excessive debility, very low temperature, age above sixty, and advanced disease of heart or any other organ. Boys take it very well in half-quantities. In Porto Rico thymol was given to several nursing mothers without bad effect to either mother or child.

If patients return to their former lives after leaving the hospital, as it is certain they will, they may become re-infected; but several old ankylostomiasis patients have returned to the medical wards for other diseases, and no eggs have been found in their motions.

As a rule I do not consider it fair to submit patients to the depressing influence of thymol more often than once a week, but in the case of fairly robust men, who were anxious to make no long stay in hospital, I have often given it every four days. Isolated cases could of course be treated with much more care than I have shown, but at Kasr el Ainy the male wards are always so full of ankylostomiasis, that we were obliged to con-

sider the greatest good of the greatest number. To prevent any possible spread of the disease, the evacuations of patients ought to be treated with some disinfectant such as Hydrarg. Perchlor., 1 in 200.

The general treatment of the anæmia is of course of great importance from the very first. The hospital mixed diet, with its meat and better bread, is superior to the food obtained by patients before admission, and contributes greatly to their improvement in health. In spite of the disturbances of the digestive tract, men, unless dying, are always ready to eat ordinary and extra meals.

Iron I have tried in various forms, and though some few patients complained of headache at first, it was greedily absorbed by the economy. My desire was to find that preparation of iron which could be taken in large doses, and would quickly banish the anæmia. The peroxide can be borne in large quantities, but the microscope proved that the blood was most benefited by the daily supply of one gramme and a half of the sulphate of iron in water in three equal doses. The patients during their stay in hospital are always taking iron, except on the days devoted to thymol. During the winter some of the feeblest patients are given every day a little brandy with advantage, but of course not on thymol days.

Digitalis given regularly strengthens the weak heart, and enables the patient to resist the effects of thymol better. I have made some experiments to try and determine, in these anæmic patients, the relative value of iron sulphate (which I usually give in a solution of glycerine and water to prevent constipation) and bone marrow in various combinations, and I have found that the greatest gain in weight occurs in patients treated with bone marrow for a week or two, followed by the iron, but here again it must be remembered that few of the Egyptian cases are not complicated by the co-existence of pellagra. If, for any reason, it is impossible to give thymol to a patient, I think the best substitute is β naphthol in three doses at two hours interval, each of one gramme.

In the mines of Westphalia, where ankylostomiasis cases have occurred to the extent of nearly 2000, distributed among 69 collieries, male fern is still employed, the routine treatment

being that the patient is given in the evening 30 cgms. of calomel and in the morning, on an empty stomach, from 8 to 12 gms. of extract of male fern, followed by another 30 cgms. of calomel in the afternoon. On the third day a third dose of calomel is given if required, and on the fourth day the male fern is repeated in the morning and the calomel in the evening. If, during the next six days, no eggs are found the patient is discharged from hospital, but if ova are present, the whole course of treatment is repeated. These large doses of male fern are sometimes as dangerous as thymol, and a fatal case occurred at Lyon a few years ago after a dose of 15 grammes, besides neuro-retinitis followed by blindness, in Westphalia. Male fern is more costly than thymol.

Patients who arrive at the hospital in a dying condition can sometimes be made to rally by saline injections per rectum, and intra-muscularly.

PREVENTION.

The Ankylostomum carrier is dangerous to the community, even if he has no symptoms.

Cleanliness alone is required, but it will be years before education will teach the fellah to wash his hands and vegetables or to protect his feet and legs. Manson suggested that men who have to work on contaminated ground should dip their feet and legs in Barbadoes tar, which is a mineral oil rich in paraffin and very cheap, and then walk across fine sand or saw dust, thus forming every morning an impervious sandal and stocking. This had already been tried in the West Indies, the idea having come from Germany where goose farmers by this method provide their birds with a protective sandal which enables them to be driven many miles to market without injury to their feet!

Latrines are the crying want of Egypt, where everything is left to the sun, and where every man eases himself wherever he thinks fit. Eleven years ago I proposed that villagers should be compelled to dig trench latrines outside their villages, to use latrines alone and to cover them daily with a little earth to deprive the embryo and larva of fresh air, but this has not yet been attempted. The best plan of all would be to disinfect

or burn the fæces. Individuals must be warned to avoid infection by keeping the hands while at work from contact with the mouth, by washing the hands and nails before each meal, and by avoiding the drinking of polluted water. The Government, in engaging recruits for the Army, Police and for Public Works labour, might have all peasants examined more carefully than is at present done for the early symptoms of this disease. Early cases of anæmia can be recognised by a doctor before the patient is aware that he has a definite illness, and one dose of thymol can prevent the chronic pernicious anæmia which drags the man to the hospital, one, two, or three years after he begins to feel ill.

The Government should also take steps to prevent this disease being introduced into the mines in the Sudan.

I am convinced that nearly all the anæmia of adult males among the lower orders in Egypt is due to ankylostomiasis, if we except those cases which are obviously caused by some other kind of hæmorrhage or by wasting disease.

The eggs in fæces can be destroyed by (1) burying them in the ground half a metre deep, provided there is no running water there, (2) exposing them for one day to dry sun heat, (3) mixing with an equal quantity of Hyd. Perchlor. 1-200, (4) mixing with a quantity of water ten times the bulk, or (5) exposure to artificial heat at 45° C. or to freezing point.

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PELLAGRA

Synonyms.—Pell' agra is the Italian equivalent for rough skin.[1] In Italy and other European countries the disease is called by many names such as "poverty illness," "sun disease," "the malady of spring sunshine" etc. In Egypt the eruption is called by the peasants *qushuf* which means chapping by the action of cold wind and water, and more rarely *gofar*, which is a term used for a skin disease which attacks camels. The condition of melancholia or dementia into which chronic cases sometimes get is called by the natives *lahooh*.

Definition.—A chronic, endemic, non-contagious, cerebro-spinal disease of poverty-stricken peasants, induced by the toxic action of diseased maize. It produces changes in the brain, spinal cord, tongue and intestines; sometimes ends in melancholia and dementia; and causes, chiefly in the spring months, an eruption on those parts of the skin exposed to the sun.

History.—Pellagra has been known in Spain since 1735, following on the introduction of maize from America about 1700. About 1750 it was found in Italy, towards 1820 in France, and about 1840 in Roumania. Since 1856 it has been endemic in Corfu, and of later years has been reported from Hungary (1888), Russia and Portugal. The case of Corfu is worth citing because so long as the peasants grew their own maize and dried it well they did not suffer from pellagra, but when vine growing replaced the maize crop, the peasants lived on bad maize imported from Roumania, which was damaged in its water transit by the Black Sea and the Danube, and then pellagra began. (Creighton).

In 1893, I began to suspect the existence of this disease in Egypt, for I found that many of the ankylostomiasis patients at Kasr el Ainy showed a symmetrical eruption which they

[1] The name was given to the disease by Frapolli, of Milan, in 1771.

believed to be of no consequence and merely due to sunburn, chapping and dirt, and moreover that some suffered from dementia and loss of knee jerk. There was at that time no one in Egypt who was familiar with pellagra, but some of my Italian colleagues on being invited to see some selected cases agreed that they resembled pellagra as seen in Italy. I then determined to visit that country in order to study the disease, and soon saw there more than 100 cases in special retreats, lunatic asylums and the insane wards of the general hospitals. I found that the disease itself was identical in Italy and in Egypt, though the Egyptians showed skin lesions over a wider area of the body, and the complications of the disease were considerably modified by the different habits of the peasants in the two countries. Since 1894 pellagra has been recognised as one of the diseases of Lower Egypt and I have seen more than 1000 patients suffering from it.

I have taken some trouble to try and find out whether pellagra was known in Egypt at any time before 1893. I find that Pruner, under the heading of "Leproses," wrote "pellagra is sporadic in Egypt, and such as we have studied it in Milan. We have seen three cases of it among the peasants, one of whom presents to-day, 12 years after our first seeing him, paresis of the upper limbs, with retraction and muscular atrophy". Dr. A. Figari Bey wrote of venereal disease appearing in Lower Egypt, with "a kind of leprous pellagra", and one or two other writers have occasionally referred to a pseudo-pellagra which may have been the real malady.

The only other Egyptian reference which I can find is a chapter in Arabic on pellagra, evidently translated from a European book, by Ahmed ibn Hassan, who was one of the first Cairo students of medicine ever sent to France.

DISTRIBUTION.

In Europe it is known in Northern Spain, Northern Portugal, the south-west of France, Northern and Middle Italy, South Tyrol, parts of Austria-Hungary, Roumania, Bessarabia, and Corfu. In some districts of Italy more than 50 per 1000 people are pellagrous, and the number who died of this disease in

Italy in 1903 was 2648. It is calculated that there are about 60,000 sufferers there of whom about 3,000 are in public institutions. In Roumania in 1899, 20 per 1000 of some districts and 7·3 per 1000 of the total population were reckoned to be pellagrous.

I have always thought it strange that I could hear no tidings of the disease from India, where there is a large population feeding on maize, and where many of the predisposing factors exist. However, at the Tropical Section of the British Medical Association meeting at Edinburgh, I had the opportunity of showing pictures and photographs of pellagra, which were independently recognized by three members from India as representing a disease which they had often seen among out-patients, without knowing its name.

In Africa it is common in Lower Egypt, but is rare in Upper Egypt, on the Red Sea coast and in Algiers. It has not been reported from South Africa, but I saw two cases there among the coloured lunatics on Robben Island in 1900. In America it exists in Mexico, the original home of maize, but is hardly known in the United States, where the poorest coloured folk live more luxuriously than the peasants of Italy or Egypt; one case has lately been reported from Georgia.

It has not been found in Australia yet, but Dr. Neiret has reported it from the island of New Caledonia.

The geographical range depends on the food supply and the poverty of the inhabitants. I imagine pellagra can be found in any part of the world where bad maize is the chief diet of the poorest classes.

This is an analysis of the homes of 490 cases admitted to Kasr el Ainy during the four years 1897—1900.

LOWER EGYPT.		UPPER EGYPT.	
Qaliubia	119	Giza	70
Menufia	98	Beni Suef	6
Daqahlia	32	Minia	11
Gharbia	20	Fayûm	2
Sharqia	44	Assiut	20
Behêra	3	Girga	5
Cairo	51	Qena	3
	367	Sohag	6
			123

These figures show the great preponderance of the disease in Lower Egypt, where maize is much grown and eaten. If we except Giza, which is the province nearest Lower Egypt, we see that the disease is rare in Upper Egypt, where the peasants live chiefly on millet (Sorghum vulgare), and not on maize.

The greatest number of cases naturally come from the provinces nearest to Cairo, and it is probably the long distance from the hospital which accounts for the paucity of patients from Behêra. It must be remembered that the poorest patients come scores of miles on foot to the hospital, begging their way. Pellagra is said to be rare in the Fayûm, where the natives eat little maize. Old students have assured me that it is rare in the neighbourhood of Luxor for the same reason, but I have seen several cases there, both in hospital and among beggars.

In 1891, after a famine, Surgeon-Captain Myles, fresh from seeing my patients, found four cases of pellagra among 3,000 starving Arabs at Tokar, on the Red Sea. The Cairenes admitted for the disease are generally beggars. In Alexandria, Port Said, and Suez, the disease is very rare, and it is not known in the Sudan, because maize is not eaten south of Korosko.[1]

About 40 pellagrous insane are admitted every year into the lunatic asylum at Abbassia, besides other insane patients who show signs of pellagra. During 1903, pellagrous insanity caused 10·5 per cent of admissions, and occasioned 10 deaths; seven of the pellagrous patients were criminal lunatics, most of whom had already committed murder before being sent to the asylum.

Pellagra is so little known among the fellaheen that I thought it would be interesting to see how far the men actually at work in the fields show early signs of the disease.

On Sept. 29th 1901, ably assisted by Dr. Talaat, I had the opportunity of examining the men and boys of three villages near Zagazig. Of 139 examined, 67 showed eruptions, tongues partially denuded and sometimes other symptoms, 14 were doubtfully affected, and 58 seemed to us quite healthy. Practically in these villages half the male population on that day

[1] I see however at the Berber Agricultural Show in April 1903, the Sudan Development Company exhibited some maize grown locally.

was pellagrous. Many of the men were suffering from advanced anæmia (probably Ankylostomiasis) and favus of scalp. At the same time I was able to examine in these villages 13 clerks and servants who did not work in the fields, and whose pay allowed them a more liberal diet; every one of them, and their employers, were free from any trace of pellagra. Again, in June 1902, by the kind help of Mr. Gibson, I was able to examine 315 men living in 11 different villages in the province of Gharbia; 114 of them, or 36 per cent., showed signs of early pellagra. In one village, where the inhabitants are especially well-to-do because they get regular pay throughout the year from the Domains Administration, there were only 15 per cent of pellagrous men, while among the men of the village which has the reputation of being the poorest, the percentage rose as high as 62. Yet all these men stoutly denied that they had anything the matter with them and the overseers in charge of them stated that the pellagrous could all do a fair day's work. On the same day I examined 23 young girls, who were working at the threshing floor or picking eggs off the cotton plants, and again I found that 30 per cent of them were already pellagrous. If these figures are in the least typical of the state of the inhabitants in Lower Egypt it is quite time that something should be done to disseminate information on the subject and gradually teach employers of labour and peasants that the early symptoms of the disease can be easily recognized, and that they will then yield to treatment, but that, if neglected, the symptoms are of a progressive nature and will end in insanity, or in general debility which entirely unfits the individual for work.

CAUSES.

Age.—I have not seen cases under the age of five years, and it is quite rare under ten. This is, perhaps, due to the fact that women do not give "polenta" of maize-flour to their infants, as is done in Italy. I am not converted to the Italian belief that pellagra is hereditary, excepting in the way that alcoholism is now believed to be so.

Out of 437 of my hospital patients 344 were men between

20 and 40 years of age. The disease, therefore, attacks adults at the very time when their health is most valuable to the State. Patients above fifty are rare, probably because they die of concurrent diseases or drift into secondary dementia.

Sex.—Nearly all my patients have been males, but this is partly due to the fact that girls and women from the country districts are not sent to Cairo. In Italy the number of women is at least equal to, if not greater than the men. I have shown that in some villages of Lower Egypt pellagra can be found among the young women.

Dr. Warnock's paper based on 141 cases at the Asylum included 49 females.

Nationality. Almost all the cases are Egyptian, for though the Sudanese and Nubians in Lower Egypt sometimes eat the same food as the fellaheen, they do not habitually work on the fields or lead the same life. Moreover, some of the predisposing causes, dirt, favus, ankylostomiasis and bilharziosis are rare among the Sudanese and Nubians. I have seen a few cases among the poorest Greeks and Turks.

Occupation.—At least 80 per cent of hospital cases are fellaheen, the others being mostly masons' labourers, boatmen, brickmakers, potters, pedlars, readers of the Koran and beggars. They are almost invariably men of the poorest class, who have been out of regular work and therefore deprived of good food for some time.

Season.—The time of year when the eruption is first noticed to appear is not necessarily the time when the patient is first poisoned, but I found on examining 300 patients that two thirds of them stated that their skin lesions were first seen during the months of January and February when the maize is very plentiful and the weather is damp. During the dry summer months there are comparatively few new cases. Some of the most intelligent patients have told me that if the eruption begins with them in February it leaves them in May, if they continue to work, but that it disappears much earlier if they rest.

It has often been noticed in Italy that pellagra is more rife after a damp summer than after a dry one, or after any failure in the maize crop.

The essential causes of pellagra are a continued diet of bad maize, poverty, and exposure to the sun; of these three, *maize* is the most important, and it is necessary that it should be diseased, and also one of the chief articles of diet. Healthy maize varied with other food will never produce disease, as may be seen by its absence among all but the poorest classes, and also among town dwellers, thousands of whom eat good maize regularly as one article of diet.

As regards *poverty* the following concrete case is a fair sample—A patient from the Fayûm, whom I saw in 1903, said he always ate millet and he picked out from several samples the kind he ate habitually. But on cross-examination, I found that he ate millet during 8 months only in the year, while for 4 months, from August to November, he ate a mixture of millet and maize, because the millet was then rare and therefore more expensive. He was in the habit of buying his food in the market and, when shown specimens of maize *(Durra Shamy)* which had been somewhat eaten by weevils, he said he would buy such maize in the market for his own consumption, but when I showed him some very bad maize covered with fungus, he said he would refuse it. This man, aged 30, only earned from 2 P. T. (5 pence) to 3 P. T. (7½ pence) in the day. On this pay he had to feed and clothe himself, his wife, and two children, but he had nothing to pay for his hut or for water. His only other food besides millet or maize was onions, buffalo milk, radishes, olives and cheese, but once a week he had a meal of cooked vegetables with a little meat.

The *sun* is certainly responsible for some of the eruption on the skin, but not apparently for the other symptoms of the disease.

Maize is grown in nearly all countries where the temperature does not reach freezing point. There are at least 300 varieties of it, some ripening in two months, others requiring seven months. The pellagra zone on maps is as nothing compared with the maize area, but the pellagra map of the world would be found to coincide with the map of the countries where diseased maize is eaten by the poorest folk. Diseased maize is often weevil eaten, but there is no evidence that these insects have anything to do with the production of the disease. The granary weevil (*sitophilus granarius*) inhabits maize stores, mills,

and threshing floors and is about one-third to half a cm. long. The female pierces a hole in the grain by means of her snout, and inserts an egg, the larva, when hatched, feeds on the grain and turns into a pupa within the seed. The complete cycle, varying from three weeks to three months, depends on the warmth of the temperature, but in tropical countries five or more broods may be produced in a year. The adult weevils also feed on maize. The rice weevil is rarer, smaller and is capable of flying, and it also feeds on maize.

There are two kinds of *durra* eaten in Egypt, one of which has apparently nothing to do with the production of pellagra; this is great millet or Guinea corn, called in Egypt by the following names; durra beledy, hiwegy, sefy, shitawi (sown in November) and nabary. This is botanically known as *sorghum vulgare* and is an annual cane-like corn, growing with a dense head of spikelets bearing numerous small grains. This durra is the chief cereal of Upper Egypt and the Sudan and it is also eaten in Lower Egypt, where it has probably been a native for hundreds of years. *Durra Shamy*, also called *durra Nili*, is unfortunately named as if it was a Syrian variety of the above named durra, it is *zea mays* or Indian corn, and is an annual, cane-like grass, which bears a dense head of closely packed grains as large as peas enclosed in a sheath. With the exception of rice, no other cereal is so widely cultivated in the world. It was introduced into Egypt from Syria about 1840 A.D. Of the different kinds of maize I find the pellagrous patients live chiefly on 14 varieties, the most favoured being *nab el gamal*, *seneibra*, *hadary*, and *moraly*, but most patients eat in addition a mixture of other kinds of maize, millet and wheat. Maize is sown in Lower Egypt in July and August and the crop is ripe in November and December. The maize crop of the Delta was calculated in 1902 at nearly 900,000 acres, being grown over about one-fifth of the cultivable land. The maize crop of Upper Egypt was about 100,000 acres, of which nearly one half was grown in the Fayûm, representing about one-twentieth of the land under cultivation. The most careful peasants never cut their durra before the sheath is dry and, after cutting it, some remove the "kusb" from the sheath as soon as possible, but the greater proportion of them keep the cob in the sheath to protect the grains from weevils, it is then exposed, heaped up on the ground, for a month on a *gurn* (the special spot for drying corn). The villagers grind the grain every few days as it is wanted, and mix it with an equal quantity of white flour to make bread. As maize contains almost the same percentage of nitrogenous matter as soft wheat and four times as much fatty matter, it stands in a high position as human food. Farmers and their servants eat healthy maize almost daily and never suffer from the disease, but the poorest peasant, who lives from hand to mouth,

and either has the habit of buying the cheapest maize in the market or selling the best of his own crop and keeping the worst cobs for his home consumption, becomes in time an easy victim. It should be noted that in Lombardy and other parts of France the maize is now very carefully dried and no pellagra exists. The poorest peasant of Lower Egypt becomes pellagrous because he lives on bad maize, while the equally poor inhabitant of Upper Egypt or the Sudan, living under very similar conditions, daily eats millet (durra beledy) and escapes pellagra. Experiments upon men, dogs and poultry have shown that pellagrous symptoms can be produced by feeding them with damaged maize or with an alcoholic extract prepared from it.

Storage.—By taking notes of 160 patients, I found that 37 of the poorest bought their maize in the market as occasion arose, while 78 kept it without the sheath, 38 with the sheath, 5 as loose grains, and 2 as ground flour. By far the greatest number store it as heads of grain with or without the enveloping sheath. To keep it inside the sheath seems the easiest way of courting damp and disaster and this is considered bad practice by some of the fellaheen themselves. It is also known to be wrong to store the heads before they have been thoroughly exposed to the sun to get dried. But, whether they keep the cobs with or without the sheath, they have very primitive methods of storing the maize during the early months of the year when rain may come and dew is always present. The heads are either kept in small mud cupboards on the ground with no sunlight and no protection from the mud floor, or in small attics on the roof equally dark, but not equally damp. Both these methods are warranted to cultivate fungi.

Preparation.—I have also taken notes of how the pellagrous patients eat their durra. Two-thirds of them eat it in the form of bread while others have it roasted and all eat it at times in the green state; this last is probably not dangerous. The habit of making Italian *polenta* does not seem to have found favour in Egypt.

As a rough and ready test of whether the durra is fit to eat one may take some from a store, crush it, damp it, and expose it to the air. I made some experiments of this kind in October 1901 with durra which was then eight months old. I found that samples taken from the barn of a Bey smelt horribly after five hours, while some other durra from a fellah's attic in the same village did not smell till after 21 hours. The following is the method employed (in Menufia province) for the preparation of bread from durra. The grains are roasted for 24 hours in an oven the temperature of which must be as high as the hand can bear, they are then taken out in a "kaffa" made of palm tree leaves, the grains are then kneaded by the bare feet in order to crush the small harsh points attached to them, they are then passed through a sieve, ground into flour at the mill and afterwards returned to the house where the flour is again passed through a fine sieve. The flour is then mixed with water in a big earthenware vessel, yeast is added, and after three or four hours the dough is baked in the oven.

Since 1903 Ceni, working in Italy, has considerably increased our knowledge as to why bad maize produces pellagra. He and others have found in cultures of bad maize the *aspergillus fumigatus*, *aspergillus flavescens* and two kinds of *penicillum glaucum* (blue mould). The first named occurs chiefly during the warm months in Italy and is by far the most important, the second one is found in Italy chiefly between October and April. The animals fed on cultures of blue mould all died, but the spores were never found to have entered the intestinal wall nor to have spread into the organs of the victim.

It is quite possible that pellagra is the result of a mixed infection and that its symptoms vary as one or other of these germs predominate. The *aspergillus fumigatus* flourishes best in a temperature of 38° to 42° C. but it lives for at least 25 minutes at 85° to 90° C. which is quite as high as that of any oven used by the fellaheen; it is killed at 100° C. If a culture of bad maize is injected into the peritoneum of animals and poultry they die and A. fumigatus is found in their lungs. Moreover, it was found in 21 out of 28 pellagrous corpses in the pleura, pericardium and more rarely in the meninges, liver and spleen. It was also found in one case in the mesenteric glands of a woman. An extract of this toxin injected into animals produced primary combined degeneration of the crossed pyramidal tracts and Goll's columns similar to the pellagrous changes seen in man. This aspergillus has been found in the fæces of pellagrous men and in the fæces of poultry fed on bad maize and after death in the lungs and intestines of the poultry. It enters the intestine in the form of spores and produces toxin poisoning in the body. Lastly, there is a sero-therapy proof, for if a goat or horse be immunized with the A. fumigatus, the serum has been found within ten days to cause improvement in the physical and mental symptoms of pellagrous patients, unless the disease is too far advanced. For further information on this interesting subject I must refer readers to the original papers of Dr Ceni and his co-workers. We seem for the first time to be within an appreciable distance of knowing why damp maize covered with fungus produces pellagra while dry healthy maize is an excellent diet.

Varieties.—There are seven kinds of pellagra known to the

country folk in Italy all of which I have often seen in Cairo, though the second and third are the most rare and the fourth is not common. (1) Those who go mad, (2) those who are drawn to water, (3) those who go backwards, (4) those who are doubled up, (5) those who become giddy, (6) those who are always hungry, (7) those whose skin peels.

SYMPTOMS AND COURSE.

It is difficult to fix any *incubation* period because the onset of the disease is so insidious. But it is perhaps worth referring again to the fact that the maize crop in Lower Egypt is harvested in November or December and that the bulk of the patients seem to begin their eruption in January. It is unlikely that the November crop could become so poisoned by the fungus as to produce a skin eruption in January after, presumably, an intervening period of premonitory symptoms. I think we must, therefore, assume that the eruption is the result of poisoning from the previous year's crop or, in other words, that incubation is perhaps from nine to twelve months duration. The *premonitory* symptoms are anæmia, failing appetite, dyspepsia, general debility, and indefinite pains, but it is difficult in Egypt to know which of these prodromata are due to pure pellagra or which are caused by the ankylostomiasis anæmia which so often accompanies it. When the disease is once established the symptoms can be conveniently divided into those affecting; (1) The skin, (2) Digestive system, (3) Spinal cord and (4) Brain.

The *skin* eruption, although the least important of the various symptoms, has given the disease its name, and has always received an undue amount of attention. It begins as an erythema, not unlike a severe sunburn and, being confined to the parts of the body which are exposed to the solar rays, it used to be thought that there was no such general disease as pellagra, and that the skin changes were all due to the action of the sun. But I hope to show that the disease should never be diagnosed by the eruption alone, and that it is no more a disease of the skin alone than leprosy or syphilis. Erythema usually appears for the first time during the spring months, because the ultra-violet rays of the sun are then specially strong. These are

evidently more productive of the eruption than the calorific red rays. The erythema when first seen consists of redness, swelling and tension of the skin for a few days, while in severe cases there is a real dermatitis producing hypertrophy with a gradual loss of elasticity. The erythema dies gradually away during the summer, so that only traces can be found in the autumn and winter, but in the spring the eruption returns with redoubled vigour and in the second or third spring the dermatitis is so extensive as to lead to pronounced exfoliation which, when present, is very characteristic.

The only rashes worthy of being photographed are seen in the spring months between March and May or sometimes in September or October. It must be remembered that in Egypt the months intervening between summer and winter are practically a second spring, tempting flowers to reappear and trees to send forth new shoots. The regular symmetry of the eruption is very striking for the erythema or icthyotic mottling extends up the forearms and legs to exactly the same level and finishes in the same crescentic way. The affected skin on the limbs is almost entirely confined to the extensor surface, though in advanced cases there is also roughness on the flexor side of the legs and on the flexor surface of the wrists. Certain exceptional cases of asymmetry prove that the rash is dependent on exposure to the sun for if the patient's occupation compels him to sit or stand asymmetrically the eruption will appear on those parts of the body which are exposed. When exfoliation begins the epidermis slowly falls off in flakes, coloured gray or brown, leaving a clean surface behind, sometimes in the form of small patches but more often as striæ, which can be well seen in Plate II. Every year the erythema leaves a little more pigment and loss of elasticity behind it until a chronic atrophy takes place marking out the sites where the eruption has been. The affected skin of a man of 30 looks wrinkled like the hand or neck of a man 80 years old, although healthy skin in the neighbourhood is still plump and elastic. Early cases complain of burning or tingling in the skin but later ones only suffer from itching. In advanced cases there is sometimes a curious icthyotic mottling, which seems to be the legacy of many attacks of exfoliation, besides emaciation of the skin with a

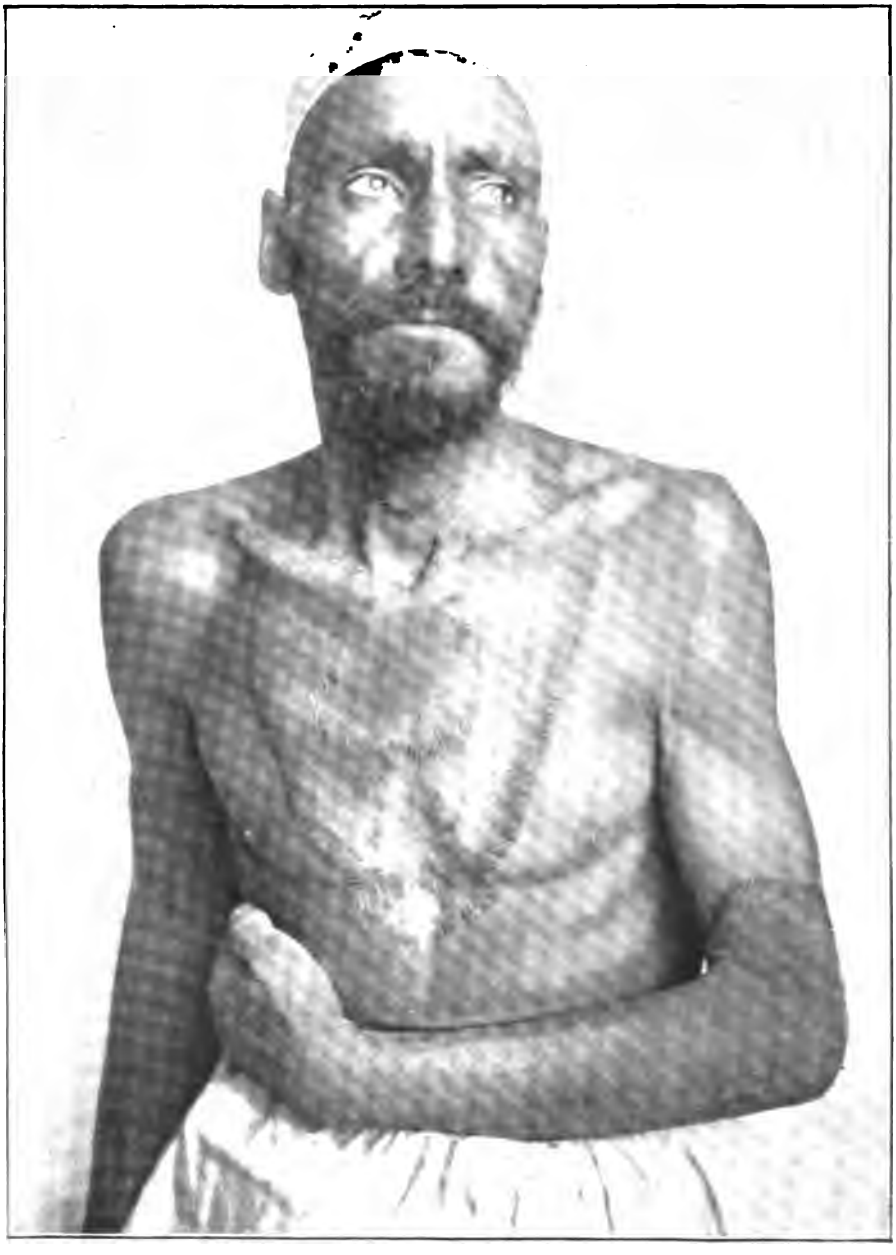
deposit of pigment and atrophy of both skin and subcutaneous tissue. The eruption is essentially dry and looks as if it was caused by the closure of the sweat and sebaceous glands; in fact, in many cases of youths, the sebaceous glands of the face are seen to be completely blocked with yellow gray matter giving the nose especially and the rest of the face a very odd appearance. Bullæ on the legs are so rare that I have only seen two or three cases. The site of the rash is limited by the patient's clothing. Adults in Europe working in the fields, get it chiefly on the face, neck, ears, and the backs of the hands, while those who habitually work with the forearms exposed get it there, those without sleeves have the upper arms affected in addition and those who go barefooted are attacked on the extensor surfaces of the feet and legs. If the Egyptian, working in the fields, wears nothing but a pair of short drawers, all the exposed portions of the body will be affected. But even in such cases the parts earliest attacked with erythema are the hands and forearms, elbows, feet, insteps, legs and knees and the upper part of the chest. In some parts of Lombardy, where the peasants worked with their clothes open at the back, erythema was seen along the vertebral column. (Bouchard). The rash shows a marked preference for the dorsal aspects of the upper and lower limbs, only creeping round to the flexor side as the case becomes chronic, and never involving the ham, groin, axilla, bend of the elbows, palms and soles. The dorsal aspect of the middle phalanx of the fingers is affected in long-standing cases, but the ungual phalanx is only rarely involved; this immunity is not, I think, due to distribution of nerves or blood, but to the fact that the worker, in grasping his hoe, seldom exposes the ungual phalanx to sunlight. The nails and hair in this disease are never affected. Every case of pellagra showing affection of the skin has patches of slight roughness on some of the extensor surfaces such as the elbows, insteps, knees, and hands. These rough patches get caked with dirt and look black or dirty gray, but if these dark coloured patches, which the patients call "qushuf" and think of no consequence, are thoroughly cleaned with turpentine, you will find, when the dirt is all removed, that there is a definite roughness on the site of the former eruption. Here

as elsewhere the original hypertrophy of the skin, due to inflammation, disappears and a gradually increasing atrophy takes its place. The only covered parts of the body which become hard and rough are the skin over the coccyx and great trochanters. I am not quite sure about the cause of the former but the trochanter spots are, I think, due to pressure by lying on the hard ground which the sick man is soon reduced to.

Case 1.—(Plate II.) A. M. æt. 36, an Egyptian, was admitted into Kasr el Ainy hospital on May 18th, 1897, with very well marked pellagrous eruption on the face, chest, nape of the neck, hands, forearms, lower third of arms, knees, legs and dorsal surface of feet. He suffered from insomnia, early melancholia, and tenderness on pressure at both sides of the cervical and lumbar vertebræ. After 25 days treatment, he was discharged looking and feeling contented, sleeping well, without any obvious eruption, with no tenderness in his back, and having gained 12 lbs in weight. The improvement in his condition was unusually rapid.

The interesting breast plate shown in this photograph is not rare among pellagrous peasants, for they usually wear a loose robe which is open at the sternum and costal cartilages. The eruption on the forearms and arms was exactly symmetrical. The portion of skin over the trachea is usually unaffected because it is protected from the sun's rays while the man is stooping at his work. The eruption, whatever its form, is greatly modified by stay in hospital and good diet, slight cases entirely disappear and severe cases lapse into a slight roughness only. About a quarter of the cases leave the hospital without any sign of roughness. A month of treatment will produce a marked improvement in a man who is desquamating extensively with brown, rough, scaly flakes of epidermis falling off his chest, face, neck, arms and legs. Judging by 100 cases, which I specially investigated with regard to this point, I find that the parts of skin are affected in this order; hands and forearms, elbows, feet and insteps, legs and knees, upper part of chest and, last of all, shoulders, neck and face. As the case becomes chronic the rash creeps round to the flexor sides of the limbs and often makes a bracelet three inches wide above the wrist, but the flexor side is never so much affected as the extensor. The

PLATE II.



colour of the eruption cannot, of course, be shown by any photograph. The normal colour of the skin of the Egyptian peasant's body may be described as *café au lait*, while the eruption is a noticeable plum colour turning into sepia as the desquamating flakes become dry and dirty. The atrophy of skin to which I have referred is best seen at the side of the neck or the back of the hands or feet and, when present, apparently means that the patient has suffered from pellagra for three or four years. Some young boys have quite wrinkled faces which make them look like a monkey or a mummy. The hypertrophy of the skin can be well seen in the neck and hands of peasants who are still at work, or of chronic pellagrous lunatics at the asylum during the spring months.

(2) *Digestive System.*—Examination of the tongue will often help to decide a doubtful diagnosis for, in most of the patients, it is more or less denuded of epithelium and papillæ and in some of the worst cases the palate also is bare. In early cases it is only the extreme tip and sides which, on examination, show preternatural cleanliness. This is one of the symptoms which improves rapidly in recent cases, though in chronic cases the improvement is very slight. The tongue is merely an index of the stomach which is very early affected, as shown by the usual evidence of chronic gastritis, epigastric pain and tenderness, flatulence and thirst. Vomiting is not present and patients often clamour for solid food when they are perfectly unable to swallow or digest it; we must therefore here assume that the appetite is exaggerated. Constipation or diarrhœa may occur quite early in the attack, and diarrhœa is an almost invariable accompaniment of the last stage. The urine shows no marked abnormality. Bleeding gums are only seen among some insane patients who have not had a free supply of vegetables. A few (4 to 10 per cent) of the worst cases of "bald tongue" show also painless enlargement of both parotid glands, but the submaxillary and sublingual glands are not similarly affected. This chronic parotitis is bilateral, never ends in suppuration, disappears if the tongue regains its epithelium, and is due, I presume, to dryness of the buccal mucous membrane and either obstruction of Stenson's duct, or the passage of bacteria into it from the mouth.

By far the greatest number of pellagrous patients have excellent teeth and perfect powers of mastication. Untreated pellagra invariably causes loss of weight and eventually produces great emaciation. Patients with diarrhœa and melancholia may lose as much as 19 lbs weight in hospital in three weeks before death, while convalescents during that period gain from five to thirty pounds. The diarrhœa is believed by Babes to be due to irritation of the sympathetic ganglia and the plexus of Auerbach. One melancholic boy of 18 lost 11 lbs weight in hospital because he refused to take his food and indulged in a religious "zikr" for 72 hours at a time!

Case 2.—A fellah, aged 35, from Gharbia was admitted on Feb. 12th 1903, with general debility and emaciation due to pellagra. He was too weak to sit up in bed and passed in the 24 hours from three to five liquid motions without control. His tongue was pale and denuded and he had pain in the back on both sides of the vertebral column from the 8th to the 12th dorsal vertebræ, the pain being greater on the right side than on the left. He was suffering from early melancholia, his knee jerk was quite absent on the left side, and almost absent on the right. He had the remains of an eruption on the back of the hands, elbows, sternum, and nape of the neck, and the skin was atrophied in all these places. He gradually improved in hospital.

The following case under treatment at the same time shows a marked improvement in weight. A pellagrous girl aged 20 was admitted for diarrhœa on Feb. 11th 1903. She had eight liquid motions and when she was discharged on April 11th 1903, her weight had risen 25½ pounds from 65½ to 91 pounds. This improvement was effected on ordinary hospital diet without cod liver oil or other fattening food.

(3) *Spinal Cord.*—If seen in the acute stage, an intelligent patient often complains of pains in the back and in severe cases this may even compel him to walk with his body arched. On examination he will be found to have pain on both sides of the dorsal column over the spinal nerves if gently pressed with the knuckle. Melancholic patients will often deny the existence of any pain in the past or present, though old cautery or seton marks near the mid-dorsal region are present in seven

or eight per cent of the cases. By examining 178 patients I found that exactly one-third of them, being quiescent cases, had no pain or tenderness on pressure when admitted to hospital, but that in 23 per cent there was general pain along the lumbar and dorsal vertebræ. In seven per cent there was tenderness between the 1st and 4th dorsal vertebræ, twenty per cent flinched between the 5th and 8th, ten per cent between the 9th and 12th, and seven per cent had pain near the lumbar vertebræ only. The most likely spot for tenderness is between the 4th and 9th dorsal spines and the tenderness is often asymmetrical, i.e. in about ten per cent of the cases. Tenderness in the back is one of the symptoms which is almost certain to disappear if the patient remains a month in hospital; about 85 per cent lose this symptom entirely, but 15 per cent will have a little tenderness or flinch at the firm pressure of the knuckle at the end of a month's treatment. One boy, aged 13, was cured though he had a history of being obliged to walk with his head bowed down because of the pain in his back. On admission he had great tenderness on the right side at the 10th dorsal, and on the left side from the 7th to the 11th dorsal vertebræ, he had cautery marks as large as a penny over the 2nd dorsal and to the left of the 9th and 10th dorsal spines. The actual cautery is a very favourite treatment among peasants for internal pain of any kind.

The knee jerk is only normal in about two per cent of the pellagra patients admitted to Kasr el Ainy. In 46 per cent the reflex is very brisk indeed, in 29 per cent it is slightly increased, in 9 diminished and in 14 it is absent. The knee jerk may also be asymmetrical, corresponding with the unequal tenderness in the back, in early cases it is slightly exaggerated, then markedly so, probably associated with early changes in pyramidal tracts. This is the time of tenderness on both sides of the dorsal and upper lumbar vertebræ and the abdominal and epigastric reflexes are then extremely brisk. Later in the case the knee jerk becomes diminished and eventually quite abolished, apparently manifesting a lesion of the posterior nerve roots in the second, third, or fourth lumbar segment. The patient, until he becomes bedridden, does not develop contraction of the lower limbs or paraplegia. He has no bed sores and

he retains control of bladder and rectum until the final stage. The knee jerk becomes less abnormal while the patient is under treatment if there is an improvement in the other symptoms. Ankle and wrist clonus are very rare, only occurring in a few incurable cases where the knee jerk is much exaggerated. There is no special *gait* in early cases but when the disease has become advanced the patient walks with the legs well apart, the shoulders raised and bent forward, and when he has reached the penultimate stage he cannot take more than a few short feeble steps without falling down, while in the last stage of all the patients are unable to stand up, or even raise themselves up in bed, and this paresis is sometimes accompanied by tremors of the limbs. (Warnock).

The final stage of pellagra is called by the Italians "tifo pellagroso" or the "typhoid state" with diarrhœa, dementia and absence of knee jerks.

(4) *Brain and Mental Symptoms.*—As of course many pellagrous patients are quite sane I propose to give under this heading only the ordinary symptoms which may be seen in a general hospital, deferring to the section of complications those mental phenomena which can be more conveniently studied in a lunatic asylum. The *facies* of the patient when he has suffered two or three years from pellagra is sufficiently characteristic; even if there is no rash present, and as yet no emaciation, one can sometimes suspect pellagra from the patient's expression. The forehead shows two sets of furrows, the horizontal ones produced by the frontalis muscle which acts from above and pulls up the skin, rather more on each side than in the middle. These furrows indicate prolonged anxiety and mental worry. Then there are the vertical lines, (well seen in Plate II.), over the root of the nose caused by the action of the corrugator supercilii or the orbicularis palpebrarum. The oblique puckering of the eyebrows increases the appearance of grief, and in some advanced cases the eyebrows have the appearance of almost trying to hide themselves inside the margin of the orbit.

Insomnia is an important nerve symptom coming on very early and remaining until the patient is given complete rest and good food, when nearly all learn to sleep all night without

assistance. Among the early brain symptoms are great mental depression without feelings of illness and discomfort, and an unreasoning discontent with everything. All thinking and calculating is an effort, the patient gradually becomes irritable and excitable with others, and stupid and morose when alone. A settled gloom is present on the face after the first year or two and patients lose all power of smiling and laughing and some refuse even to try to smile; when eventually they are induced to make the effort, they make uncouth noises and facial contortions showing they have literally forgotten how to use their risible muscles. During the first two years of pellagra, in an individual of average intelligence, no definite mental symptoms can be noticed, but after that he is found to be always sad, decidedly stupid, and unwilling to take interest in anything beyond his food and sleep. His hopeless eyes stare vacantly into space, he loses his memory and becomes distinctly melancholic, always discontented, craving incessantly for food and cigarettes; yet he loses weight in spite of being granted extra food. The sleeplessness now becomes more pronounced and there are vague feelings about the head, sensations of weight, pressure, emptiness or pulsation. There is often a vague purposeless restlessness culminating in a fixed, unreasonable desire to get away from the place in which he is. From this stage, it is not difficult to get to a further one of resistiveness, refusal of food, and suicidal tendencies. There is evidently a connection between the burning and itching sensations in the skin when affected and the frequent delusions of being burnt, of sorcery and persecution; some patients think their skin is being stretched or pulled off, and that they can escape from their tormentors by plunging into the Nile. Hallucinations of taste and smell are frequent and the gastric troubles lead the patient to refuse all food and to imagine every one is poisoning him. His delusions are in fact insane renderings of his subjective symptoms.

There is all this time a gradually increasing tendency to mental depression, the downward course, from discontent to sadness, melancholy and then confirmed melancholia, ending, in the incurable cases, in secondary dementia. At least half of the pellagra patients who apply for help at a general hospital

are already melancholic though of course very few of them are as yet insane.

Case 3.—A man called by his friends "little John the dumb" was admitted to Kasr el Ainy on Aug. 21st 1895, in a stuporous condition, refusing to speak or answer any questions. I sent him to the lunatic asylum on Dec. 12th 1895, where he remained a little more than a year, when he was returned to Kasr el Ainy where I again kept him till May 3rd 1897. On leaving hospital he was in a state of secondary dementia, but had gained 20 pounds weight, had learned to speak and could be utilized for some of the menial services in the wards. His chief treatment consisted of intermittent electricity, cod-liver-oil, strychnia and other tonics, enforced exercise in the garden, massage and salt baths for half an hour twice a day.

Case 4.—M. H. An Egyptian peasant woman, aged 30, was admitted to Kasr el Ainy on April 17th 1897. She had a well marked pellagra rash on the arms and legs, her tongue was denuded, she was extremely thin and weak and abnormally hungry. She passed her excreta in bed or anywhere in the ward, and had to be prevented from eating dirt. She was melancholic, unwilling to talk and, when spoken to, she repeated the question and seemed unable to reply. At night she would get out of her bed and walk about the passages. She had favus all over her scalp, and ankylostomiasis which required four doses of thymol. We afterwards found from her relations that one morning she had gone down to the river to bathe and had then wandered some miles along the river bank until she reached Cairo. On Oct. 9th she was discharged from hospital having increased in weight 32 pounds, from 81 to 113 pounds, "she now smiles, talks, helps the other patients in the ward, understands all that is said to her and seems quite sensible, she has no eruption and her tongue is normal, but her knee jerks are still too brisk and she still weeps rather easily if she has any disagreement with the other patients."

The temperature of an uncomplicated case is always normal or subnormal.

The whole course of the disease may extend over five, ten or even fifteen years.

COMPLICATIONS.

There are certain constant accompaniments of pellagra in every country: extreme poverty, insufficient food stuff for constant labour and a general neglect of all hygienic laws. But, in addition, there are several varying factors which weigh down the unfortunate sufferer.

In Italy the pellagrous adults are often addicted to alcohol and are also victims of uncured syphilis and malaria, accounting for some of the special complications discovered by Italian writers. In Egypt, alcohol is not a vice of the peasants, but syphilis and malaria may both occur, and an almost constant accompaniment of pellagra is ankylostomiasis, sometimes joined by bilharziosis; other entozoa, and favus of the scalp are often present, and the patient succumbs eventually to tuberculosis of the lungs or abdomen. The ankylostomiasis anæmia probably predisposes to the degeneration of the spinal cord. Wrist-drop, paraplegia, general tremors, ataxy, epileptic seizures, retention of urine, herpes zoster, bronchitis, and kidney disease are sometimes present.

Insanity.—By Dr. Warnock's courtesy, I have been able to see many of his pellagrous lunatics and have taken notes of some of their delusions. One man thought his hands were being burned and that he had a devil in his belly, which told him to leave the hospital or he would die at night. Two months later he was cured of his delusions, had increased 9 pounds in weight and had been rid of 631 ankylostoma worms by thymol.

Another man said that for two years he had been possessed by a devil, who lived just above his umbilicus, and always told him what to do.

Another said he had a devil in his body and limbs, and spent the day in begging the other lunatics to cast the devil out of him, while another said that there were people who were trying to lift him up from his bed through the ceiling to the roof. He was nearly always silent, and dirty in his habits, and carefully enquired whether my stethoscope was an instrument to kill him. Another case (published by Dr. Warnock), aged 20, of a dwarfed, shrivelled appearance kept repeating the statement that he had drunk poison and would be killed. He died at the asylum 2½

years after admission; "a restless urgent melancholic, distressing his neighbours by his cries, catching hold of visitors and imploring protection from his poisoners, he often refuses food and declares his abdomen to be full of poison; he is usually sleepless, declaring that when he sleeps his enemies fill him with poison, he steals food when unobserved and suffers from diarrhœa, he has back pain between the scapulæ, and his tongue is raw looking." The form of insanity occurring with pellagra, seems to be one peculiar to it, not simply the result of malnutrition and anæmia of brain. Many criminal lunatics brought to the Cairo asylum are found to be pellagrous, they have been arrested for some purposeless murder in consequence of delusions which they forget and deny a few days after admission. One such case was said by his family to be quiet generally but to have violent outbreaks every winter. He remained melancholic but quiet at the asylum for eleven months and then suddenly (in Jan. 1904) attacked another patient without warning, and would have killed him if the attendants had not interfered; his excuse was that the man was beginning to persecute him.

There is another type of pellagrous insanity which deserves special mention. Instead of melancholic ideas, the patient develops expanded notions of himself and has exaggerated sensations both mental and physical; although emaciated and unable to stand, he declares he is in perfect health and extraordinarily strong. The differential diagnosis of a case of this type from general paralysis is often puzzling, especially when the skin lesions have disappeared, and former eye disease makes it impossible to note the reaction of the patient's pupil.

Case 5.—(published by Dr. Warnock) A peasant, aged 45, was admitted from Gharbia to the asylum on July 1st 1901, delirious, talking nonsense, destructive and dirty in his habits. He had no signs of syphilis, was emaciated and anæmic, and had a typical pellagrous black rash on the back of the neck, legs, forearms, hands and feet. His gait was paretic, he staggered and fell after a few moments and while standing kept his feet widely separated; his knee jerks were exaggerated and he had well marked ankle clonus. His tongue was fairly steady, and though his speech was slow, his articulation was good. He had cautery marks along his spinal column and tenderness

on pressure over the dorsal vertebræ, he gradually became worse and, when quite bedridden, he still smiled and said that he was very happy, as strong as ten men and called himself the "lion of lions." He was childish, passive, prostrate and demented, with much impairment of memory. He died on Sept. 25th. and at the P. M. twelve hours after, well marked pachymeningitis hæmorrhagica was present. "The dura mater was thickened, and on its under surface, over the superior and lateral surfaces of the left hemisphere there was a layer of brownish red semi-transparent membrane adherent to the dura mater but peeling easily, and on the under surface of the dura mater were numerous rusty stains. The pia mater and arachnoid were healthy but there was some milky opacity over the 4th ventricle. These membranes stripped easily from the convolutions and left *no* erosions. The vessels at the base were healthy; the brain was generally soft and flabby and collapsed on the table; on section no obvious changes were seen except anæmia, excess of fluid and marked dilatation of the lateral ventricles. The heart was in a condition of brown atrophy, the intestines were very thin and contained ankylostoma worms."

For the next case, ending in recovery, not yet reported, I am also indebted to Dr. Warnock.

Case 6.—An Egyptian woman, aged 21, was admitted to the asylum on May 15th 1904, with a history of pellagra for the last six months. She was married seven months before admission, but her husband had divorced her because she wandered about for no reason, was sleepless, performed the religious "zikr," talked to herself and used to fall down when she tried to walk. On admission she was sleepless, pulse 120, tongue could not be seen, she had pellagrous rash on her elbows, legs, and trochanters, and her knee jerks were greatly exaggerated. Her expression was very dull, she complained of being ill in her body and stated that she was possessed by a devil. On the same day she had a sort of fit during which the attendant stated that she had contraction of the limbs and head for a short time but no loss of consciousness. She could walk, but refused to stand up when asked to do so and was quite demented, forgetful and unable to converse rationally. She was excited at times, incoherent and noisy, but was able to sup-

port herself if she grasped something to pull herself up by. A month later she was still restless, always talking, dirty in her habits, but had no definite delusions. In July, she still had staggering gait, was subject to falls and her muscles were contracted apparently involuntarily, her brother, who then visited her, said that she had had a black rash on her face and hands that "would not wash off." Her brother denied any syphilis in the family, but had evidently himself suffered from pellagra. In August she was still childish and dirty in her habits, still obliged to catch hold of something to support her, when standing up, and her speech was still defective, especially the labial sounds, but she was more cheerful and beginning to put on weight. In November she was fatter, quiet, but still childish and her speech still defective, she was able to walk and inclined to do some work. She laughed inanely and volunteered that she had been under sorcery induced by a man in her village, but the effects had now passed off. In December it was noted "does some work and has some sense but is shy and imbecile in demeanour." In January, eight months after admission, she had improved so much that she was able to be discharged, quite strong physically and able to work, but mentally still somewhat childish, still thinking she had been under sorcery. Her weight increased in the asylum from 121 to 132 pounds.

Many symptoms in the lunatics resemble general paralysis of the melancholy type in the last stage, and I have seen several patients in the asylum who were thought probable cases of general paralysis until pellagra was diagnosed, either by their bodily and mental symptoms quite clearing up, or by the development of a new pellagra rash. I should here state that in the asylum maize is never an article of diet so that it is impossible for these chronic cases to get re-infection while under treatment, however, it is interesting to note that some of the chronic pellagrous lunatics in the asylum, spending several hours a day in the sunshine develop a new erythema every spring, which is absent at other times of the year. The mental symptoms do not show themselves until the 3rd or 4th year of the disease and their late appearance suggests that the insanity is caused secondarily by malnutrition of the brain, anæmia, and general cachexia and not directly by the toxin; but on the

other hand early dementia, early loss of memory and childishness point rather to organic brain disease.

Mortality.—The percentage of my Kasr el Ainy cases gives 18 cured, 72 relieved, 6 unrelieved and 4 deaths.

PATHOLOGY AND MORBID ANATOMY.

At an autopsy there will at once be seen great emaciation and cachexia, generally with marked anæmia. There may be definite exfoliating patches on the parts of the body exposed to the sun during life, or there may be only a little roughness on those parts, but the skin there, if carefully examined, will be found to be atrophied, and there is a general diminution of subcutaneous fat. Microscopically there is sclerosis of the blood-vessels, papillæ, and corium, as well as atrophy of the horny layer.

The muscles, heart, liver, spleen, and kidneys share in the general atrophy.

The lungs sometimes show tubercular lesions. The stomach reveals no lesion to the eye, but the walls of the intestines are thinner than usual, and show a slight shedding of the superficial layers of the epithelium, with atrophy of the muscular tissue. There is no ulceration of the intestines. Many naked-eye lesions have been reported by various observers as occurring in the brain, but the only constant one is atrophy of the cortex of the convolutions, especially the frontal.

I have taken to England one or two brains and cords which Dr. Mott has kindly examined for me, and though they did not arrive in a condition which permitted of thorough examination he tells me that he has been able to find in them evidence of chronic slight but diffuse meningo-myelitis.

The spinal cord shows no decided change until it is prepared and carefully examined. Tuczek in 1893 found in eight autopsies in Italy that all of them showed symmetrical sclerosis of the columns of Goll. In six cases there was also lateral sclerosis in the dorsal region, and in one case he found cervical anterior sclerosis.

In 1894 Pierre Marie contrasted pellagrous sclerosis with the posterior sclerosis of tabes dorsalis. In 1899 Dr. F. E. Batten

kindly made for me many sections of three pellagrous cords, and furnished me with a report upon them. The following notes refer to the case which provided his sections as seen in Plate III.

Case 7.—An Egyptian woman aged 33 was admitted under my care on Dec. 18th 1898, complaining of diarrhœa, general debility and pain in the lower extremities.

She was extremely emaciated, and weighed only 67 pounds. The symptoms of pellagra were: slight roughness and atrophy of the skin on the dorsal aspect of the hands and feet and also on the back, a very denuded tongue, diarrhœa, and motions passed in bed, dementia with persistent muttering, absent knee-jerks, some sleeplessness, and tenderness on both sides of the spine near the 3rd dorsal, and 1st, 2nd and 3rd lumbar vertebræ.

The diarrhœa was somewhat checked in hospital, where her mental condition required bromide and chloral. The temperature was practically normal throughout, but her weight fell persistently to 61 pounds, and she died on January 23, 1899, twenty-six days after admission.

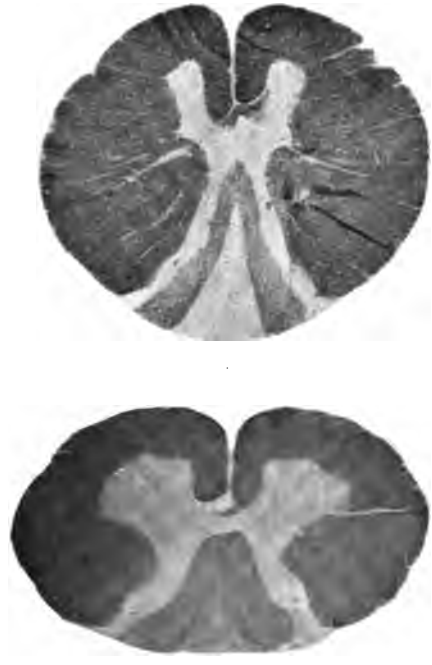
No ankylostoma eggs were found in the fæces, though she was extremely anæmic. The urine contained a distinct trace of albumen. At the post-mortem examination there was slight atheroma of the aorta; the heart weighed 185 gms.; the lungs were congested and œdematous, but otherwise normal; the liver weighed only 790 gms.; the spleen 140 gms.; the kidneys each weighed 70 gms., and were markedly cirrhotic; the brain weighed 1050 gms., and was very œdematous, while the membranes of the vertex were thickened and opaque.

The spinal cord, when examined in London, showed marked changes.

"1. *Marchi's method.*—The paleness of the posterior columns was very noticeable, but under the microscope no recently degenerate fibres could be seen. The cells of the anterior horn were pigmented.

2. *Weigert-Pal method.*—The lack of fibres in the posterior columns was very marked, both sacral and lumbar regions being affected equally. In the mid-dorsal region a pair of normal roots entered the cord, and wedged itself in between the atrophied fibres of the median and external columns; this root

PLATE III.



Sections of pellagrous spinal cord showing posterior sclerosis of Goll's and Burdach's columns. Both sections are from the same cord, the dorsal (above) shows the sclerosis better than the cervical (below).

could be traced up to the upper cervical region, where again the incoming roots contained more normal fibres. A small wedge-shaped tract was also visible, just outside the anterior horns in the cervical region.

3. *Aniline-blue-black method.*—The increase of the connective tissue in the posterior columns was very marked, and distributed itself in exact correspondence with the condition of the roots above described, namely, a pair of roots which had undergone no degeneration in the dorsal region showed no increase of the connective tissue in the area it occupied in the cord. There was no increased vascularity of the cord, the cells of the anterior horn and the nucleus and nucleolus were distinct, the increase of the connective tissue was limited to the posterior columns except in the wedge-shaped tract above described in the cervical region, which appeared darker owing to the smallness of the film in this area.

4. *Van Gieson's method.*—There was no evidence of any recent inflammatory action in the grey matter. There was some thickening of the walls of the smaller vessels, especially in the posterior columns, though it was not limited to this region.

The posterior roots of the cervical, dorsal, and lumbar regions were also examined in this case by Marchi, Weigert-Pal, and Stroebe's methods. Marchi's method showed very little recent degeneration, though it was obvious, from the lack of staining, that a very considerable amount of degeneration had taken place, and this was made evident by staining by the Weigert-Pal method. The greatest amount of destruction seemed to have taken place in the dorsal and lumbar regions, and to a lesser extent in the cervical region; the same condition was also shown by the Stroebe stain; only a few axis cylinders could be seen in each root."

This patient died of pellagra and chronic kidney disease, but there was no possibility of knowing for how many years she had suffered from pellagra.

The cord degeneration would appear to be of root origin and affects the extra-medullary, as well as the intra-medullary portion of the posterior roots. The degeneration in the cervical region of this cord was most marked in the columns of Goll, the columns of Burdach being affected to a lesser degree.

Since then I have had many other sections cut for me which have been examined by experts, but unfortunately nothing of pathological interest was revealed. The absence of cord degeneration in these cases was due to the fact that the patients had either had pellagra for too short a time, say one year or less, or that, though they had suffered from pellagra for three or more years, the clinical signs of the disease were not very far advanced. In other words, spinal cord degeneration as discovered by the microscope is a comparatively late lesion in the disease.

Experiments on poultry.—Lombroso's experiments proved that birds, when fed on damaged maize, could die in a few months of acute pellagra, and further researches have been repeated since 1899 by Dr. Ceni in Italy. He finds that when poultry are fed on bad maize they show the first symptoms of the illness at the beginning of the spring season. The symptoms decline in the height of summer, sometimes recur slightly in autumn, and again remain quiescent during the winter. This autumn recrudescence in Italy is interesting, because it corresponds with the autumn eruption sometimes seen in men in Egypt. The symptoms in poultry are: diarrhœa, which is sometimes bloody, gradual loss of feathers which in some cases is almost complete, a characteristic erythema of the skin, and rapid emaciation, causing some of the birds to lose more than half their own weight. During the second half of summer the birds gradually improve, lose their diarrhœa and eruption, and regain to some extent their weight and feathers, so that towards the end of November their appearance is normal, in spite of the continuance of the same bad food. The hens die at various periods between one and four years, and it would seem probable that those which live longest are more likely to show symptoms similar to chronic pellagra in man. The weight of the hens before death declines from about 1700 to about 810 grammes.

At autopsies of the acute cases, there is general atrophy of the muscles, congestion of all the internal organs and chronic enteritis. In some cases there is, in addition, an acute adhesive fibrinous pericarditis. A culture made from some of these cases in Raulin's solution showed *Aspergillus fumigatus*.

At autopsies of those which die of progressive debility, after three or four years of the damaged maize diet, there is a greater atrophy of muscles, complete absence of subcutaneous fat, and a characteristic thinning of the skin. The atrophy is especially noticed in the intestines, where there is also chronic diffuse enteritis.

Chickens hatched from eggs laid by pellagrous hens, show no more resistance to pellagra than chickens resulting from healthy hens, but poultry fed for a long time on bad maize proved easily susceptible to the pathogenic action of the spores of the Aspergillus, though this is not the case with healthy poultry. The spores of the Aspergillus would seem to be very dangerous, for if a very small quantity of them are injected into the pericardium of a hen, they cause death from acute or subacute pellagra, and at the autopsy the spores are still found in the pericardium, surrounded by inflammatory cells.

In Egypt it is the custom to give to the poultry durra which is too bad for human consumption.

Diagnosis.—The physician is hardly likely to see the early erythema or the thickening of skin on the affected parts, because the patient postpones coming to hospital until he has had the disease for two or more years and he is beginning to find he cannot work. On careful examination and removal of dirt by baths and turpentine, there will probably be found the dry scaly condition of the extensor sides of the limbs, extremely symmetrical, but distributed only over those portions of the face or body which are exposed to the sun, and the skin in the affected regions will generally be found to be somewhat atrophied in comparison with the normal skin elsewhere. If the patient is made to dress in the clothes in which he works in the fields it will be seen that the principal sites of eruption exactly correspond with the parts exposed to the sun. The typical case presents no difficulty because it is the only known disease causing this special group of symptoms: dermatitis on exposed parts of the body, a denuded tongue, sleeplessness, altered knee jerks and tenderness on pressure in the dorsal region. The disease can only be diagnosed with certainty in the poorer peasants and beggars who live chiefly on maize. The peasant will often deny that

he eats maize or that he has a skin eruption recurring at the beginning of the year, but he will confess to certain marks on his skin which he says are due to dirt and the action of the sun and wind. There will perhaps be dark indurated patches on the elbows and knees. *Sprue* patients suffer from obstinate diarrhœa and denuded tongue, but I have never seen this disease among Egyptian peasants. No one acquainted with psoriasis or leprosy will be likely to confound pellagra with them. In *ichthyosis*, as in pellagra, there may be an accumulation of blackened scales which crack in the direction of the lines of the skin into lozenge shaped pieces, again, the soles, palms, and flexures of the limbs are the parts in both diseases in which these defects are least noticed, and there is a distinct absence of secretion of both sweat and sebaceous glands. But it must be remembered that ichthyosis is a congenital maldevelopment of the skin dating from the second or third year of life, and that the skin of the face is seldom affected. In pellagra, on the other hand, the eruption is very rare under the age of ten years, and almost always attacks the face.

Prognosis.—Early cases can be cured by giving tonics and generous diet without maize. The dangerous symptoms are obstinate diarrhœa and insomnia, total loss of the knee jerks, increasing emaciation, suicidal melancholia or dementia and the concurrence of tubercle. The disease may last many years in spite of the accompaniment of ankylostomiasis and bilharziosis.

Treatment.—Fresh air, rest, ablutions and a liberal diet of meat are the first essentials. Even sound maize should be excluded from the diet for fear of unconsciously adding to the poison. The patients will often ask for extra bread; and for loss of weight cod liver oil is very useful. Fresh bone marrow is a distinct help to some of the worst cases, and one can judge of the general improvement by gain in weight and increased sleep. There is no satisfactory drug treatment for the disease, but strychnia or arsenic will be found the best tonics. If there is any suspicion of malaria quinine should be given, and as almost every individual is an early or late case of ankylostomiasis the thymol and iron cure is obviously indicated, in order to benefit the pellagra and anæmia at the same time. Baths and zinc ointment will help the skin eruptions to disap-

pear and improve the patient generally. Insomnia may require opium or sedatives, and the diarrhœa will tax every effort of food and drugs. Opium should not be given if kidney disease is present. I have tried massage and electricity for some of the melancholic patients without much benefit. Excited and suicidal cases will require extra food, sedatives and special watching. Since 1889 attempts have been made by Babes and others to find an anti-toxic serum.

The *popular* treatment hardly exists because the disease is not yet known. The patient is generally taken by his friends to the village barber who burns with hot irons any painful part of his body such as his head, epigastrium or back. If that does no good, setons are tried, and a journey is made to a mosque or sheikh's tomb to pray for recovery, last of all he applies for relief to the Government Hospital, or is brought there by the police, starving and homeless, and very often unable to answer questions intelligently.

PREVENTION.

In Italy much good has been done since 1890 by providing some of the pellagrous districts with improved means for drying, storing and baking maize, and by establishing special farms and retreats for the care of patients. But the Egyptian government has so far been unable to initiate any reforms of this kind. The disease is so wide spread in Lower Egypt that it would be impossible to make pellagra a notifiable disease, but much might be done to teach the peasants the difference between good and bad food. Sound maize flour damped and kept at a temperature of 25° to 30° C. will remain sweet for more than 24 hours, while the diseased flour will give off a disagreeable odour in less than eight hours. The peasants should be taught the necessity of sowing better maize, for diseased seeds seem to produce a dangerous crop. A remedy employed for killing weevils in the grain might be tried as an experiment against the *aspergillus fumigatus*. Bisulphide of carbon is a clear, colourless liquid of strong odour which evaporates quickly and leaves no smell. 1½ pound of it is enough for a ton of maize in a storehouse, and cotton saturated

with this liquid can be placed just below the surface of the grains so that the heavy vapours which come off may descend into the heap of corn and kill the weevils. If some method of this kind were regularly employed it would no longer be necessary for the peasants to keep the cobs in their sheaths as a protection from weevils.

It has long been known from experience in India and other countries that, when seed infested by the spores of fungi has been sown, the blight has been reproduced in the crop; this is true of rust, smut and bunt which all affect both millet and maize, and experiments should now be made to see if the *aspergillus fumigatus*, like them, is reproduced in the new crop, and if so, whether the seeds should not as a matter of protection be dipped in some solution before being sown.

I have ventured to recommend three measures to the Government:

1. That the village authorities of Lower Egypt (mudirs, omdehs, sheikhs, etc.) should be informed by the usual methods of the Ministry of the Interior, that although good maize is an excellent food, the habitual use of bad maize produces a disease affecting not only the skin, but also the digestive and nervous systems. The question is now of increasing importance, for *durra shamy* is much more cultivated than it was a few years ago in consequence of the increase of the population and the more bountiful supply of water. On the other hand the wages of the fellaheen have nearly doubled during the last fifteen years and therefore they can now afford better food including more meat.

2. Maize is sold at the weekly market of every town; it is stored in "shunas" in all large towns, and it is, in addition, sold as a surplus stock by the fellaheen from their own land to their neighbours. It seems to be impossible to control or inspect the sale of maize, but I think the local authorities should be informed that it is improper to allow obviously diseased maize to be exposed for sale. But it is not the worst maize which finds its way into the market, for the worst samples cannot command a price. It is the poorest peasants who are the chief offenders, for at the end of each year their custom is to take, in lieu of some of their wages, a piece of land, which they cultivate for themselves with a crop of maize; the

most careless of them sow diseased seed, gather the crop before it is ripe, store it in damp places before it is properly dried, and habitually eat the worst cobs which they cannot sell.

3. Maize is imported into Egypt from America, and also from Syria and Roumania, both of which are countries affected by pellagra. All imported maize should be examined in Alexandria before it is allowed to be sold there. The quantity varies every year from a minimum of 109 tons in 1894, to 61,905 tons in 1898. Of all endemic diseases to which Egypt is subject I believe pellagra to be one of the most important.

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